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THE AMERICAN JOURNAL OF ROENTGENOLOGY
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CARDIAC PHYSIOLOGY REVEALED BY THE ROENTGEN RAY*

APPLICATIONS OF ELECTROKYMOGRAPHY, BIPLANE ANGIOCARDIOGRAPHY AND CINEFLUOROGRAPHY

By HERBERT M. STAUFFER, M.D., MORTON J. OPPENHEIMER, M.D., LOUIS A. SOLOFF, M.D., *and* GEORGE H. STEWART, III, B.S.
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WILLIAM HARVEY began his "De Motu Cordis" (1628) as follows: "When I first tried animal experimentation for the purpose of discovering the motions and functions of the heart by actual inspection and not by other people's books, I found it so truly difficult that I almost believed with Fracastorius, that the motion of the heart was to be understood by God alone. I could not really tell when systole or diastole took place, or when and where dilatation or constriction occurred, because of the quickness of the movement. In many animals this takes place in the twinkling of an eye, like a flash of lightning. Systole seemed at one time here, diastole there, then all reversed, varied and confused. So I could reach no decision, neither about what I might conclude myself nor believe from others. I did not marvel that Andreas Laurentius wrote that the motion of the heart was as perplexing as the flux and reflux of Euripus was to Aristotle."[12]

Similar difficulties have plagued generations of clinical fluoroscopists yet fluoroscopic observation remains the simplest and most practical roentgenologic method of obtaining functional information about the heart. Various more elaborate techniques have been developed to obtain more precise and more detailed information about this most important, complex and dynamic organ. Kymography, angiocardiography and roentgen cinematography are perhaps the outstanding special methods for such cardiac study. The accelerating progress of cardiac surgery provides a constant impetus for extending and improving these techniques.

This presentation is a brief review of examples illustrating the application of these techniques in our institution where, over recent years, a stimulating and productive

* From the Departments of Radiology, Physiology, Medicine and Medical Physics of Temple University School of Medicine and Hospital. These studies were supported in part by grants from the United States Public Health Service (No. H-2234) and from the Heart Association of Southeastern Pennsylvania.
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collaboration in this field has developed between the Departments of Radiology, Physiology, Medicine and Medical Physics.

I. ELECTROKYMOGRAPHY

Multiple slit roentgen kymography has been widely used for analyzing cardiac border motion but has many well recognized limitations. To overcome some of these difficulties the electrokymograph and its initial laboratory and clinical application were developed in our institution by Henny, Boone and Chamberlain, as a cooperative effort with the United States Public Health Service.[13]

It is of historic interest in this connection that in 1925 Chamberlain made motion pictures of the cardiac silhouette with Ruggles' roentgen cinematograph which produced 15 8×10 inch direct roentgenograms per second; the roll of film was used up in 30 exposures (2 seconds). By measurements from the resulting films, kymographic curves of the movements of various points along the cardiac silhouette were plotted showing their behavior in various phases of the cardiac cycle.[3]

When the photomultiplier tubes became available, they provided the means for detecting changes in roentgen-ray transmission due to changes in heart thickness or to movement of its border. The 931-A tube became the "pick-up" of the electrokymograph and the fluoroscope was used to localize it over the desired part of the heart. The device is capable of producing very accurate records and these are readily correlated precisely with records of other circulatory phenomena such as the electrocardiogram, the carotid pulse and intracardiac pressure. Proper interpretation of the records requires much experience and the bugaboos of positional change of the heart as a whole and of overlapping of various pulsating structures have not been eliminated by this more elegant method of kymography. Further, much of the cardiac mechanism occurs internally with relatively little reflection in surface activity.

Evidence of asynchronism of ejection from the ventricles in man was recorded by this method.[9] Analysis of electrokymograms of the pulmonary artery and the ascending aorta demonstrated that asynchronous ventricular ejection was more frequent than synchronous ejection in the normal subjects studied. Increased degrees of asynchronism were found in patients with bundle branch block.

In extensive studies in the laboratory, good correlation was found between the change in heart thickness in dogs (density change recorded by the electrokymograph) and the cardiac output determined by the Fick and Stewart methods.[21,22,23]

More recently, unpublished electrokymographic studies based on left ventricular density curves have shown a reduction in the early ventricular ejection rate in patients with hypertensive cardiovascular disease as compared with normal subjects.[20]

In the normotensive group over fifty years of age, 33 per cent showed ejection velocities which slowed early in systole (after 0.04 seconds), while 78 per cent of the hypertensives showed early slowing. In one patient with coronary artery disease, slowing of early ejection was recorded during an attack of angina with a return toward normal after administration of nitroglycerine. This patient ultimately died of myocardial infarction. Another patient in the control normotensive group, without cardiac symptoms at the time of study, showed ejection slowing and has since died of myocardial infarction also. One other normotensive with early ejection slowing has since developed angina and myocardial infarction but has survived. The early ventricular ejection rate as estimated from electrokymographic density curves may thus provide an important clue to the functional status of the myocardium.

In studies of the left atrial border electrokymogram, we were unable to confirm the observations of others that the electrokymogram of the left atrium in mitral regurgitation is pathognomonic and significantly different from the records in subjects

with physiologic mitral systolic murmurs.[25]

The considerable bibliography on electrokymography is collected in the proceedings of the conference held at the National Heart Institute in 1950.[4] The recent monographs by the Swedish roentgenologists, Andersson,[2] Nordenström[18] and Rudhe[24] attest to the continuing interest in this tool for clinical and laboratory cardiovascular investigation.

II. STUDIES OF INTRACARDIAC FLOW WITH BIPLANE ANGIOCARDIOGRAPHY

The usual clinical tests of circulation time measure streamline flow making use of extracardiac receptors. They include extracardiac flow and do not distinguish variations in flow in individual chambers. Detailed angiocardiographic studies of circulation time within the heart have been carried out in our institution[26] using the biplane stereoscopic apparatus devised by Chamberlain.[28] Exposures in two planes simultaneously have proved of value in this work in the identification of chambers and the timing of the appearance of contrast material.

The biplane stereoscopic apparatus incorporates mechanical features which provide considerable flexibility, making it useful for both cerebral angiography and angiocardiography. The stereoscopic feature has been of value chiefly in the study of cerebral angiograms but is also of aid in the orientation of intrathoracic structures which exhibit relatively little motion such as the pulmonary veins (differentiation from left atrium). Simultaneous exposures are made in each plane at intervals of 0.7 seconds with exposure times from 1/10 to 1/20 second. This exposure time is too long to adequately "stop" the more rapidly occurring cardiac motions so that blurring of contrast margins is frequently encountered.

In the studies of flow in adults, the duration and intensity of opacification of each chamber was determined in each plane of projection. The anteroposterior and lateral projections were routinely employed in this study. The roentgen exposures were correlated with the carotid pulse by graphic recording with a two channel Brush recorder. Exposures were made for a minimum of 20 seconds after the injection of 50 cc. of 70 per cent urokon. The results were plotted as density against time in a fashion similar to the method used by Lind, Spencer and Wegelius.[17] The volume of the left atrium was estimated from its dimensions in the two projections during contrast filling.

The normal intracardiac circulation time, from initial visualization of the left atrium to that of the aorta, did not exceed 6.3 seconds. The normal right heart time is less than the intracardiac time, while the pulmonary artery and left atrial times approximate the right heart time. Opacification of the pulmonary artery disappears before the aorta is visualized, as noted by Figley.[10]

The intracardiac circulation time may be prolonged in heart disease but was found not to exceed 9.1 seconds in the absence of clinical heart failure, atrial fibrillation, or marked cardiomegaly.

Using this technique, 22 patients with mitral stenosis and 31 patients with mitral stenosis who had had mitral valvulotomy performed were studied. In mitral stenosis a disproportionate prolongation of the pulmonary artery time and the left atrial time was found. The left atrial time exceeded the pulmonary artery time and the latter, in the absence of right heart failure, exceeded the right heart time. In mitral stenosis the left atrial volume was estimated as 2 to 12 times normal. However, no correlation could be found between the left atrial volume or the left atrial time and the surgically reported size of the mitral orifice, the age of the patient, or the known duration of the rheumatic state.

These angiographic findings in mitral stenosis are of importance in reference to the pathophysiology of this lesion since they fail to support the present dominant concept that left atrial changes are proportional to the degree of narrowing of the mitral orifice and to the duration of the obstruction. Similarly, there was no correla-

tion between left atrial time or volume and right ventricular time, suggesting that the left atrial changes are not the immediate cause of right ventricular alteration. All of these findings appear to emphasize the importance of the extent of myocardial disease in the dynamics of mitral stenosis.

The angiographic pattern found in mitral valvular disease cannot be considered diagnostic since it may be found in stenosis, regurgitation or both or even in the presence of nonvalvular heart disease.

Simultaneous biplane angiography appeared to be essential in the evaluation of the contrast-filled left ventricle. Significant and unpredictable differences were noted in the visibility of the contrast in this chamber in one projection as compared with that in the other projection, simultaneously exposed. Such variations are apparently due to the position of the heart, enlargement of the ventricle, and the position and obliquity of the septum. There are marked systolic-diastolic variations in left ventricular opacification while left atrial opacification appears practically constant throughout the cardiac cycle. In mitral stenosis the left atrium undergoes relatively little cyclic change in size or degree of opacification, whereas the left ventricle almost completely empties itself of contrast material in systole unless failure is present. Thus, films in very rapid sequence or a record correlating the roentgen exposures and cardiac events are mandatory.

III. IMAGE INTENSIFIER CINEFLUOROGRAPHIC ANGIOCARDIOGRAPHY

(*a*) *Persistence of Light from Intensifier Output Phosphors.* The persistence or lag of the commercially available image intensifier phosphors was investigated in connection with our interest in the use of these intensifiers in cinefluorography because it seemed obvious that such persistence must have a bearing on the problem of securing sharply detailed exposures of short duration in rapid sequence to effectively "stop" very rapid motion. The persistence of light from the phosphors is obvious to the observer when the image intensifiers are used fluoroscopically and the roentgen beam is cut off.

Studies of the persistence of light from the output phosphors were made by Dr. George C. Henny. The image intensifier tubes under consideration were lined up with a constant potential roentgen beam. Between the roentgen tube focal spot and the intensifier tube was a spring-loaded lead shutter in front of a rectangular aperture in a sheet of lead. When the lead shutter was allowed to intercept the roentgen beam, the roentgen rays were quickly stopped in a way that depended upon the speed of the shutter and the size of the aperture. This rate of cut-off was determined by first employing the same physical arrangement with a light beam acting directly on a photomultiplier, the output of which was, in turn, recorded through an oscilloscope and camera with a known time axis. The geometry of the light beam and the photomultiplier duplicated the geometry of the roentgen beam and the front screen of the intensifier tubes. The same photomultiplier picked up the light output from the intensifier tubes and recorded it in the same way. The difference between the two time versus light output curves was therefore due to the light peristence from the output phosphor of the intensifier

TIME LAG IN THE LIGHT FROM THE INTENSIFIER OUTPUT PHOSPHOR

Intensifier Tube	Time for intensity to decrease to fraction of original intensity					
	1	3/4	1/2	1/4	1/8	1/16
Westinghouse	0	.9 msec.	1.6 msec.	2.5 msec.	7.5 msec.	18.2 msec.
Philips	0	1.7	2.6	7.6	24	82

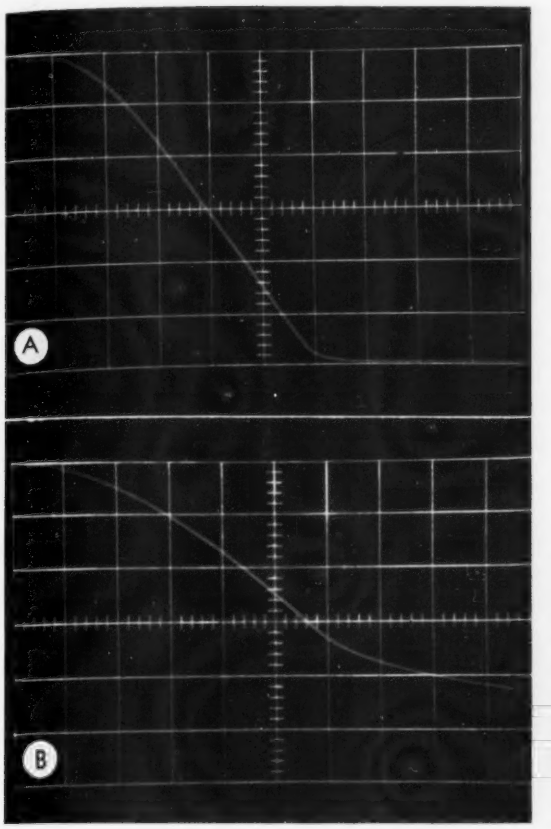

Fig. 1. (*A*) Oscilloscopic trace showing the actual movement of the lead shutter across the aperture defining the roentgen-ray beam. The height of the curve is in arbitrary units of brightness of light passing through the aperture. The vertical lines represent the time base and the spaces between the squares indicate 1 millisecond intervals. (*B*) Oscilloscopic trace of the light output of the image intensifier which was activated by roentgen rays passing through the same aperture and cut off by the same shutter as was used in *A*. The difference between the two at any moment, therefore, is an indication of persistence of light emission by the intensifier output phosphor.

tube (Fig. 1). The persistence in light output is recorded below in terms of the time required in milliseconds for the original or maximum intensity to decrease to three-quarters, one-half, etc., of this brightness.

The subsequent studies of motion blurring indicate that the initial drop in light intensity is so rapid that the prolonged persistence at low light levels becomes of little practical photographic importance.

(*b*) *Rapid Motion Blurring and Exposure Time.* Dotter and Rogers[6] and Kjellberg, *et al.*[15] have emphasized the need for short exposure duration to "stop" rapidly occurring intracardiac motions. Wegelius and Lind[29] have documented the need for rapid sequence exposures to obtain a full record of the events of the cardiac cycle particularly at rapid heart rates in infants. The most rapidly occurring events in the cardiac cycle occur at the peak of systolic ejection and in animals the velocity of the aortic blood has been estimated to range from 90 to 160 cm. per second at this time with the mean velocity in the aorta and other large arteries approximately 20 cm. per second.[5] "Streaming" of opaque contrast material thus occurs at such rates briefly, and valve structures certainly reflect these rates.

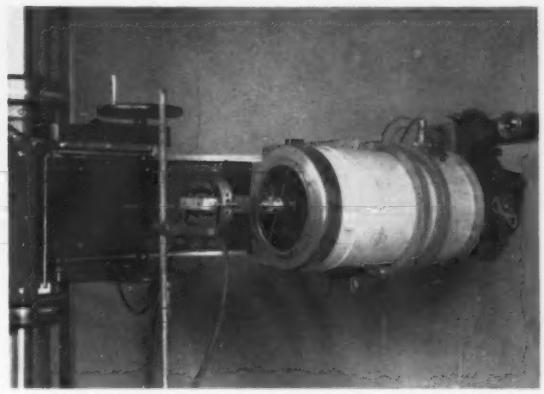

Fig. 2. Rotating test object for demonstrating motion blurring in image intensifier cinefluorography.

In order to study the use of the image intensifier in "stopping" rapidly occurring motions, a rotating test lucite disk was arranged (Fig. 2). The lucite disk, 14.6 cm. in diameter, has a square section lead strip 2 mm. by 2 mm. imbedded on the diameter. The disk is pivoted at its center and is rotated by an induction motor. The lead strip rotating about the axis, when placed in a roentgen beam, casts a moving shadow which falls on the input end of the image intensifier. The speed of rotation of the disk was arranged so that the velocities encompassed between the ends of the lead strips include the faster velocities expected in studying intracardiac events, 24 to 120

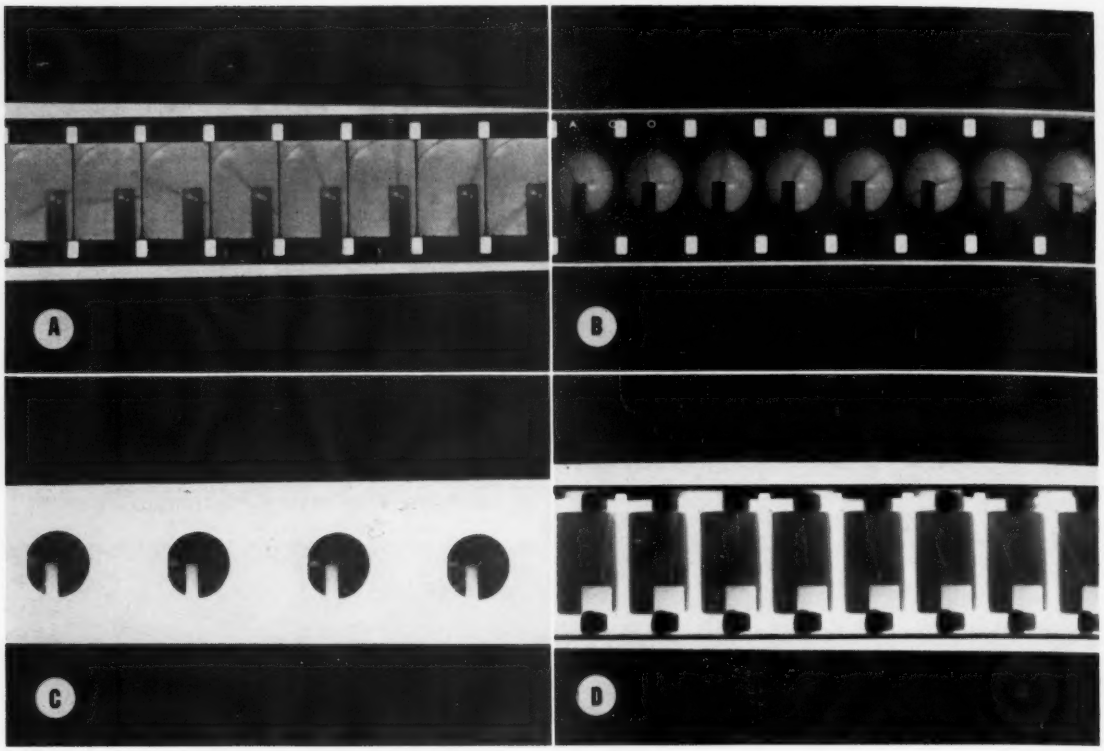

FIG. 3. (*A*, *B*, *C* and *D*) Movie frames of motion blurring tests (see text).

cm./sec. Before the experiment, the exact speed of rotation of the disk is determined with a stroblite. Pictures of the disk taken through the intensifier may show motion blurring at the outer end of the lead strip and much less or no observable blurring at the inner end. From the speed of rotation of the disk and the point along the lead strip which does not show objectionable blurring, the maximum velocity without objectionable blurring may be found for any given exposure duration.

Figure 3*A* shows the result of tests made under various circumstances with this test disk. With a standard Bell and Howell 16 mm. camera, exposures were made of the test disk in daylight at 64 frames per second (duration of the exposure per frame is approximately 1/120 sec.), and these showed marked blurring increasing toward the periphery of the disk. Blurring of similar magnitude was recorded when exposures were made through the image intensifier (Philips) at 64 frames per second (Fig. 3*B*). With full-wave rectification the cable capacitance has provided sufficient "smoothing" effect at currents up to 10 ma. so that objectionable flicker has not resulted.

Exposures were made of the same test object with the same image intensifier and optical setup with exposures of 1 millisecond duration using a high tension switch tube (Machlett Dynapulse.)* For this test the camera was run at 8 frames per second and the illustration shows a sample sequence in which the Dynapulse exposures happened to be synchronized with the movie frames (Fig. 3*C*). It will be seen that there is no appreciable blurring of the test lead strips even at the periphery of the disk.

Similar exposures were made of the test disk employing a Kodak high speed industrial camera operating at approximately 500 frames per second. For this test a constant potential roentgen-ray generator was

* Through the courtesy of Dr. Barton R. Young, Germantown Hospital, and Franklin X-ray Company, Philadelphia, Pennsylvania.

employed operating at 80 kv. and 8 ma. In spite of the fact that the phosphor was continuously illuminated in this experiment, the resulting movie frames show insignificant motion blurring along the entire length of the test strips (Fig. 3*D*). The exposure duration per frame in this experiment with the high speed camera is of the order of 0.4 millisecond.

A direct roentgenogram of the test object made with a single pulse (60 cycle current) showed appreciable blurring at the periphery.

Exposure durations of movie film of the order of 2 milliseconds per frame can be obtained with cameras having independently variable shutter apertures. For instance, with certain cameras the shutter aperture can be reduced to one-quarter, and at 64 frames per second this would result in an exposure duration of approximately 1/480th of a second. Films exposed at the $\frac{1}{4}$ shutter setting showed blurring of the test object reduced to an acceptable level.

These studies suggest that in clinical work with motion pictures roentgen exposures of 2 milliseconds (1/500 second) or less synchronized with the motion picture frames are desirable for studying intracardiac events in the greatest detail and to minimize the roentgen-ray exposure to the patient. This might be achieved by the use of a high tension switch tube. Diminishing the shutter aperture of the motion picture camera, using constant potential roentgen ray, is a simpler alternative but will be limited clinically to studies of very short duration and will find principal application in the laboratory.

The attempt to secure sharp detail of the rapidly occurring intracardiac events on each individual frame is important because of our desire to study and have available for reproduction the static roentgenographic image. However, it should be emphasized that much information can be obtained about moving structures when they are viewed in motion pictures and especially in slow motion even though the individual movie frame shows an extremely unsharp image from which little or no information can be extracted. This has been well illustrated by our studies with visualization of intracardiac structures with carbon dioxide gas where the moving valve leaflets have been well visualized in the projected motion picture despite grain and motion blurring which impair the quality of the static image.

(*c*) *Experimental Use of Carbon Dioxide as Intracardiac Contrast Material.* In the course of studies of air embolism in dogs, conventional biplane roentgenographic studies showed the possibilities of the use of gas as an intracardiac contrast material.[7] Jacobi, Janker and Schmitz,[14] and Ejrup, Lind and Wegelius[8] have also reported roentgen studies of the heart with gas contrast. Subsequently the safety of intravenously injected carbon dioxide even in large doses in dogs was shown and many cineroentgenographic observations at 64 frames per second, employing the image intensifier, were made.[27] Two infants tolerated large doses without mishap.

Since the presentation of reports on these studies, carbon dioxide injections have been made intravenously in 10 adults. These patients received 25 to 100 cc. of carbon dioxide with no subjective discomfort. In 2 of these receiving 50 and 60 cc., respectively, simultaneous right ventricular pressure determinations (via heart catheter) and the electrocardiogram showed no significant alterations. These patients were examined supine, and in films made with the biplane angiographic apparatus the outflow tract of the right ventricle, the trunk of the pulmonary artery, and the valve level were demonstrated well by the gas contrast.

Similar "selective" angiography of the outflow tract of the right ventricle and the trunk of the pulmonary artery has frequently been carried out in dogs, employing 1 to 2 cc. of carbon dioxide intravenously per kilogram with rapid injection. One of the most striking things about the resulting motion picture studies made in the lateral projection with the animals

supine is the regurgitation of gas in ventricular diastole from the pulmonary artery back into the outflow tract of the right ventricle. The bolus of gas thus oscillates back and forth with small increments being forced on each expulsion into the peripheral pulmonary circulation until the gas is dissipated. When the film is examined in slow motion, the valve leaflets are frequently well visualized. Thickening of the free wall of the right ventricle with little inward movement is observed in systole; the interventricular septum approaches the free wall expelling blood and gas.

Regurgitation of the gas through the aortic valve is observed on injection of quantities of the gas into the left ventricle and regurgitation through the mitral valve has also been observed. Eddy currents in the blood stream are believed to be responsible for maintaining the valve leaflets in place so that they close promptly when there is a critical change of pressure at the end of ventricular systole.[3] Elimination of these eddy currents may be an important contributing factor in this striking regurgitation of gas through valves which are competent for blood.

Narrowing of the pulmonary artery was produced experimentally in dogs by placing a ligature about the artery immediately distal to the pulmonic valve, and such supravalvular pulmonic stenosis has been well demonstrated by the use of intravenously injected carbon dioxide in motion picture studies.

Injections of carbon dioxide have also been made directly into the left ventricle through a catheter introduced into the carotid artery and aorta. When large doses of the order of 7.5 cc. per kilogram are injected in this manner, within a few seconds regurgitation of large quantities of the gas occurs through the mitral valve and the left atrium is visualized together with gas entering the pulmonary veins. In one instance gas also appeared in the right auricular appendage, and subsequent postmortem examination of the animal demonstrated a foramen ovale which was patent to a probe. Such a demonstration indicates a possible application of the gas contrast method in the demonstration of intracardiac septal defects.

"Double contrast" studies of the left ventricle have been done with injection of diodrast simultaneously or immediately before the injection of the carbon dioxide. The opacified blood produces excellent contrast with the gas in the heart, and the coating of opacified blood on the walls of the heart chambers and the aorta and aortic valve produces good visualization of these structures.

In one instance in which double contrast injection was made into the left ventricle the subsequent study of the motion picture film showed very dramatically bubbles of gas advancing peripherally through the opacified coronary arteries. This phenomenon has occurred regularly when gas injections were made, but we have not observed the actual bubbles of gas in coronary arteries without opaque contrast material in the blood. A radiolucent line girdling the heart was well defined in the movie studies of several dogs; subsequent coronary artery opacification showed that this represented fat in the coronary (atrioventricular) sulcus.

Many determinations of various physiologic parameters have been made in the course of the studies of the effects of introduction of carbon dioxide into both the right and left sides of the circulation. The cardiovascular respiratory disturbances are minimal when intravenous carbon dioxide is employed. The duration of most changes is of the order of seconds although a small elevation in right ventricular systolic pressure and a moderate polypnea may last for several minutes. The changes in arterial carbon dioxide, oxygen and pH are also very small after injection of carbon dioxide gas. Carbon dioxide gas also produced only minimal disturbance in circulatory and respiratory phenomena on introduction into the left ventricle or even into the peripheral portion of the carotid artery, and, here again, disturbances lasted only for a few seconds.[19]

The motion picture studies with gaseous intracardiac contrast have frequently shown valve structures well in the slow motion projected movie, whereas detail was obviously blurred, largely due to rapid motion when the static individual frame was viewed. The effect of the rapid motion of blood in producing blurring is evident when opaque contrast solutions are studied and the streaming phenomena are observed during early filling of a cardiac chamber or one of the great vessels. The study of the individual gas bolus in motion also provides a good indication of the need for exposures of very short duration in visualizing the phenomena occurring particularly during ventricular systole. For instance, a gas bolus may be visualized in the outflow tract of the left or right ventricle on one frame in sharp detail and on the subsequent frame, 1/64 second later, it may be visualized several centimeters beyond in the pulmonary artery or aorta, as the case may be, having traveled several centimeters in this time indicating a velocity in excess of 150 cm. per second. The detail of the bolus in motion during this time and of the valve structures through which it is passing is obscured in the exposures we have been using which are of the order of 1/120 second.

For these 16 mm. motion picture studies at 64 frames per second with the dog in the lateral projection and the roentgen beam horizontal, the roentgen factors have averaged 90 kv. and 6.5 ma. with 2 mm. of aluminum added filtration (Philips image intensifier). This results in an air dose of approximately 7.5 r per minute at the entrance portal. This roentgen-ray intensity was required when fine grain, high resolution panchromatic film (Eastman Recordak) was employed. For many purposes much information was obtained from angiocardiography both in the animal and in infants employing Tri-X film with reduction in the roentgen-ray intensity by a factor of 10 but with a more grainy image.

(*d*) *Mitral Competence in Experimental Left Ventricular Angiocardiography.* Because of the interest aroused in the possibility of determining mitral competence in patients by direct percutaneous injection of opaque contrast material into the left ventricle (Lehman, *et al.*[16]), we have performed left ventricular angiocardiography in dogs by catheter introduced through the aorta into the left ventricle with image intensifier cinefluorography. Figley and his associates[11] have previously reported that they were unable to demonstrate with conventional serial roentgenography evidence of regurgitation of opaque contrast material into the left atrium of dogs on percutaneous direct puncture injection into the left ventricle (1 cc./kilogram injected in ½ sec.). This was true when the mitral valve was presumably normal, but marked regurgitation was observed when a chorda tendinea was previously cut.

Of 7 dogs in which we injected 70 per cent diodrast by catheter, there was slight regurgitation evident in the motion picture studies in 4 with relatively slow rates of injection (1 cc./kilogram in 5 to 7 sec.). In one experiment there was no detectable regurgitation.

In one instance, with more rapid injection of a larger volume of diodrast (1.6 cc./kilogram in 5 sec.) and marked bradycardia (morphine-chloradose anesthesia), massive mitral regurgitation was demonstrated occurring during ventricular diastole (Fig. 4). Two catheters had been introduced into the left ventricle, one for injection of diodrast and the other for pressure recording. A pulmonary artery catheter recorded the pulmonary "wedge" pressure, a reflection of left atrial pressure. The resulting record shows elevation of left ventricular, femoral artery and pulmonary "wedge" pressures following the injection of diodrast, attributed to inspiration (Fig. 5).

Further studies have shown that these rises are secondary to the hyperpnea produced by diodrast. There is subsequent hypotension and tachycardia attributed to the peripheral vasodilating effect of diodrast.[1] At necropsy the tip of one of the catheters was found distorting trabeculae carneae in the left ventricle but the papillary

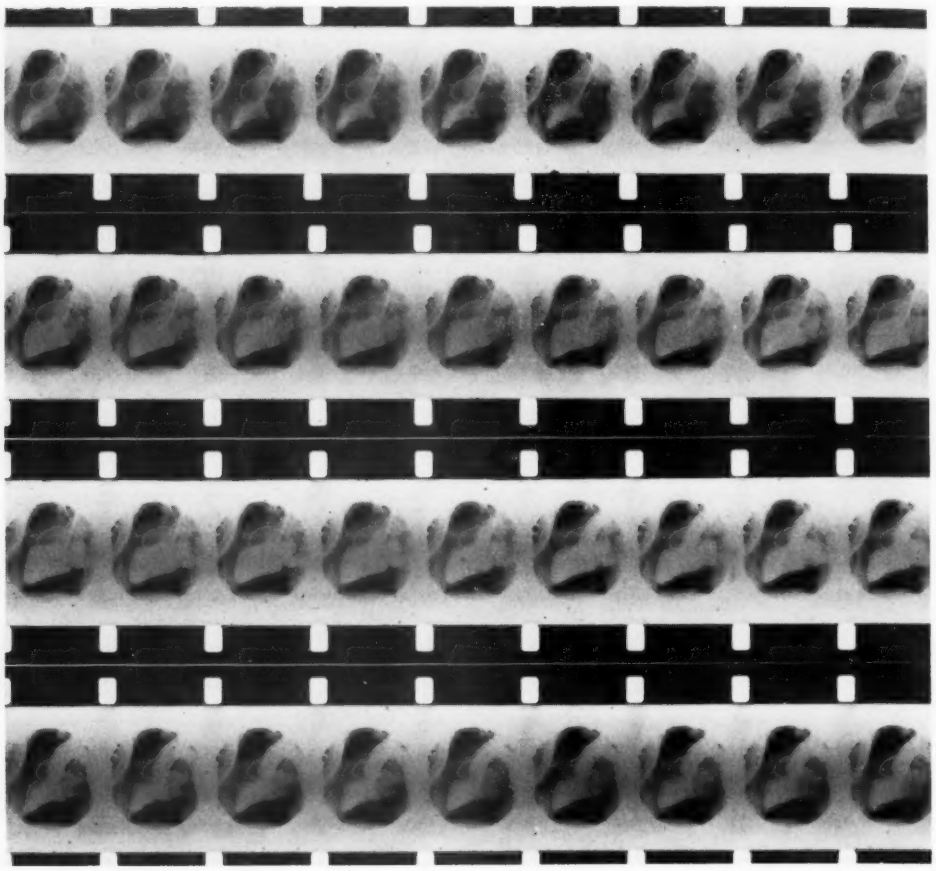

FIG. 4. Left ventricular injection of diodrast via catheter in dog. Cinefluorogram showing mitral regurgitation.

muscles and chordae tendineae did not seem involved. Although the presence of the catheters in the ventricles and the bradycardia may have influenced the cardiac mechanism, the demonstration of such marked regurgitation with an apparently intact mitral valve is of importance in evaluating clinical diagnostic studies where injection is made by the percutaneous method.

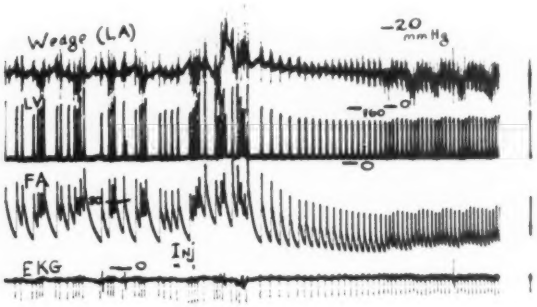

FIG. 5. Left ventricular (LV), femoral artery (FA) and pulmonary "wedge" pressures following left ventricular injection of diodrast (Inj.) shown in Figure 4. Initial pressure rise due to hyperpnea. Note subsequent marked fall in left ventricular and arterial pressures and tachycardia.

In the remaining similar experiment, without bradycardia (nembutal anesthesia), marked regurgitation occurred in systole on repeated injections. This "physiologic" regurgitation of contrast material continues as long as opacification of the left ventricle persists.

(*e*) *The Left Atrium in Cinefluorographic Angiography of Human Subjects.* The normal emptying of the left atrium in ventricular diastole with the piston-like cephalocaudad displacement of the atrioventricu-

lar ring has been well shown in image intensifier cinefluorographic studies in an infant. The film in this instance was made at 64 frames per second in the lateral projection.

For comparison, a similar study was made of the left atrium in an adult with long standing mitral stenosis and surgically produced mitral regurgitation (commissurotomy). This study showed the marked enlargement of the left atrium with prolonged visualization of this chamber and poor visualization of the left ventricle with the opaque contrast material. As compared with the normal there was strikingly little change in the anteroposterior dimension of the left atrium during the cardiac cycle with most of the observed motion of the atrium occurring in the cephalocaudad direction due to oscillation of the atrioventricular ring. This study in the lateral projection in an adult was made with the image intensifier at 32 frames per second.

SUMMARY

Examples of the application of electrokymography, angiocardiography and cinefluorography in the study of cardiac physiology are presented.

Slowing of the rate of early ventricular ejection in electrokymographic density curves may be an important index of the functional status of the myocardium.

The pattern of intracardiac flow of opaque contrast material in angiocardiography in the normal and in mitral stenosis is outlined.

Studies of light persistence and motion blurring with image intensifiers indicate that the initial rate of decrease in light intensity is so rapid that the phosphor "lag" does not interfere with the usefulness of exposures of 2 milliseconds or less per frame in "stopping" rapid cardiac motion in high-speed cinefluorography.

Cinefluorographic observations with the experimental use of carbon dioxide alone and in combination with diodrast as intracardiac contrast are mentioned.

Cinefluorography has repeatedly shown mitral regurgitation of opaque contrast following injection into the left ventricle via aortic catheter in dogs.

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CARDIAC VENTRICULOGRAPHY*

DIRECT TRANSTHORACIC NEEDLE PUNCTURE OPACIFICATION OF THE LEFT (OR RIGHT) VENTRICLE

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ROENTGENOLOGIC visualization of the chambers of the heart became a reality in 1937 with the introduction of angiocardiography by Castellanos, Pereiras and Garcia,[2] and its further elaboration in 1938 by Robb and Steinberg.[8] However, with the Robb and Steinberg method of angiocardiography certain limitations of chamber opacification are observed. Paramount among these is the fact that opacification proceeds from a systemic vein to right heart chambers, thence through pulmonary circulation to left heart chambers, which course of the opaque substance obviously results in varying degrees of overlap of chamber upon chamber, as well as progressive dilution of the opaque bolus and consequent progressive decrease of opacification.

In an effort to improve chamber opacification, some investigators introduced the opacifying agent by means of a catheter inserted through a systemic vein, with the catheter tip advanced to a location in either the right atrium or the right ventricle. Chávez, *et al.*[3] in 1947 published their experience with "direct intracardiac angiocardiography," utilizing the catheter technique.

Transthoracic needle puncture of the heart and the introduction of an opaque medium into the ventricular chambers in dogs was reported by Reboul and Racine[7] in 1933. Nuvoli[5] in 1936 reported a successful injection of a sodium iodide solution into the left ventricle of a patient, via an intercostal approach. Ponsdomenech[6] and Beato Nùñez[1] in 1951 described their experience with direct heart puncture and opacification of ventricular chambers in 30 humans, terming their procedure "cardioangiography."

Smith, Wilson, Cregg and Klassen[9] of The Ohio State University in 1954 reported 75 ventricular punctures and opacifications in 18 dogs, and also reported use of this procedure on 6 humans. These workers, too, applied the term "cardioangiography" to the procedure. They discuss possible values of direct needle puncture opacification of the ventricles, and tabulate potential hazards with suggested preventive measures. In September, 1955, Cregg, Smith, Wilson and Bull[4] further discuss cardioangiography and report experiences with the procedure in 19 patients.

The technique of ventricular puncture and direct ventricular opacification was introduced to us by Dr. Philip W. Smith** while on a visit to our institution in February, 1956, and the first 2 of our series of 77 ventricular punctures were performed by him.

Our mounting interest in direct ventricular opacification was stimulated by our need for a more adequate objective assessment of the presence and degree of mitral valvular insufficiency, and for the evaluation of surgical procedures directed toward correction of mitral insufficiency. Direct left ventricular opacification has also been used by us for appraisal of aneurysms of the left ventricle and, since excellent opacification of the thoracic aorta is usu-

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ally achieved, also in assessing certain aortic lesions. We have recorded certain observations regarding aortic valves and have noted the regular opacification of the coronary arteries, information which may ultimately prove to be of significant value. Our experience in the demonstration of interventricular septal defects has not been sufficiently extensive for critical analysis of the effectiveness of the procedure in the diagnosis of such lesions.

Direct right ventricular opacification is accomplished by right ventricular puncture, and the evaluation of tricuspid insufficiency by this procedure is the subject of a study now in progress and to be reported later.

While Ponsdomenech[6] and Beato Nùñez[1] and, later, Smith, *et al.*[9] applied the term "cardioangiography" to the procedure, we have adopted the term "cardiac ventriculography," and affix either "left" or "right," depending upon the ventricle opacified. We have continued to employ this term in the belief that it expresses more accurately the direct opacification of the ventricle and also more distinctly differentiates the procedure from angiocardiography.

Our experience with the performance of 77 cardiac ventriculographies in 60 patients is the subject of this report.

METHODS AND MATERIALS

As with other elaborate roentgenologic procedures, cardiac ventriculography requires a well-coordinated "team."

The supervision and direction, by a radiologist, of the roentgenologic component of the procedure are requisites. The necessary observations of simultaneous pressure and electrocardiographic recordings demand the full-time attention of a member of the team conversant with manometric and electrocardiographic equipment, and with the significance of the recorded pressure and electrocardiographic tracings.

The ventricular needle puncture *per se* should, in our opinion, be in the hands of a physician prepared to meet possible emergencies such as cardiac arrest.

A component member of the "team" is the anesthesiologist, prepared to administer oxygen, and to initiate other emergency measures in event of possible complications.

Premedication. Premedication of the patient is indicated to allay apprehension and pain, and to aid in preventing certain cardiac conduction disturbance potentialities of the opacification procedure.

We employ a combination of seconal or nembutal, atropine sulphate, and demerol, administered one hour before the examination. For the average adult, the barbiturate dosage is 1½ gr. (0.1 gm.). Atropine dosage is 1/200 gr. (0.3 mg.). Early in this series, patients with a pulse rate below 85/minute received atropine in dosage of 1/150 gr. (0.4 mg.). However, this larger dose resulted in atrial tachycardia in several patients. The routine use of atropine was then omitted in 8 patients. Of these 8, 4 had a short period of asystole immediately following injection of the opaque medium. We, therefore, resumed the use of atropine, employing 1/200 gr. (0.3 mg.). Demerol has been selected as the narcotic of choice because of its minimal depressant effect upon cardiorespiratory function. Adult patients weighing less than 125 pounds receive 25 mg.; patients weighing 125 pounds or over receive 50 mg.

Equipment. Rapid serial filming, during and following injection of the radiopaque medium into the ventricular cavity, is essential for satisfactory cardiac ventriculographic study. The Universal Automatic Seriograph* has proved satisfactory in our hands. For adults, 11 by 14 inch films are quite adequate, with filming at the rate of 2 per second for 6 seconds. Roentgenographic equipment with capacity of 200 ma. and 120 kv., and a heavy duty rotating anode tube are necessary. Conventionally, however, we have employed a tube current of 300 ma.

As cardiac puncture is accomplished, pressure tracings and electrocardiographic changes are continually observed by means

* Manufactured by The Automatic Seriograph Corporation, College Park, Maryland.

of a capacitance type electromanometer and a direct-writing poly-oscillograph.*

We utilize a 6 inch, short-beveled needle for the ventricular puncture. In the majority of our cases, that has been gauge 15. A gauge 17-T (thin-walled) needle was used in a smaller number of cases, but its use was associated with an incidence of glass syringe breakage in 2 cases. With improvement in syringe strength with resultant avoidance of syringe breakage, the gauge 17-T needle is preferred. The needle is connected to a 20 cm. length of polyethylene tubing, 2.0 mm. in lumen diameter, having a Luer-Lok adapter at each end and metal clamping guards at the connections of the tubing to the adapters. A Luer-Lok three-way stopcock connects this tubing with a similarly equipped 50 cm. length of polyethylene tubing. The three-way stopcock, the lumina of which have been enlarged, is inserted between these two sections of polyethylene tubing to permit connection with the electromanometer for the purpose of monitoring pressures during intracardiac needle insertion.

A syringe is attached to the terminal adapter of the tubing assembly. Early in the series we used a 50 cc. Robb syringe.** As aforementioned, we encountered occasional difficulties arising from syringe breakage during the injection procedure. We had the Robb type syringe modified with a stronger piston, and although the incidence of syringe breakage was reduced, we still experienced occasional syringe breakage. A further improvement, with an increase in wall thickness of both the piston and the syringe barrel, was devised by one of us (J.S.L.) and we have now obtained a stronger glass syringe.† Using this latter syringe, we believe our problem of syringe breakage has finally been resolved.

The syringe is adapted to a mechanical pressure injector‡ (Fig. 1), also devised by one of us (J.S.L.). Utilizing this injector, which employs a simple lever principle, we are able to deliver 50 cc. of radiopaque substance through the aforementioned tubing and needle assembly in two seconds or less.

The basic equipment we employ in the performance of cardiac ventriculography is illustrated in Figure 2.

The radiopaque medium used throughout our entire series of cardiac ventriculographies has been 70 per cent diodrast, although our experience with angiocardiography suggests to us that there unquestionably are other marketed media which are also satisfactory for cardiac ventriculography. We have found that 40 cc. of 70 per cent diodrast will effect excellent opacification of the left ventricle and subsequent good visualization of the aorta. In the study of tricuspid valvular insufficiency with right ventriculography we have, on a number of occasions, employed 50 cc. of 70 per cent diodrast, and we believe this to be the optimal amount.

For the management of possible severe reactions or complications, aside from the usual facilities of the anesthesiologist, a laryngoscope, an endotracheal tube, a thoracotomy tray, a cardiac defibrillator, and a supply of levophed, benadryl, hydrocortone, and 5 per cent glucose in distilled water are also on hand for emergency use.

ROENTGENOGRAPHIC TECHNIQUE

We have attempted to determine the optimal projections for the demonstration of various lesions. All of our cardiac ventriculographies have been obtained with the patient in *horizontal* position. An exaggerated right posterior oblique position, actually a right lateral projection with the patient's right side down but with approximately 15 degrees posterior tilting, has been our preferred projection for the demonstration of mitral valvular insufficiency with its regurgitant opacification of the left atrium.

* Obtained from Sanborn Company, Cambridge, Massachusetts.

** Becton Dickinson & Company, Rutherford, New Jersey.

† Becton Dickinson & Company, Rutherford, New Jersey.

‡ Obtainable from Franklin X-ray Corporation, 2100 Arch Street, Philadelphia, Pennsylvania.

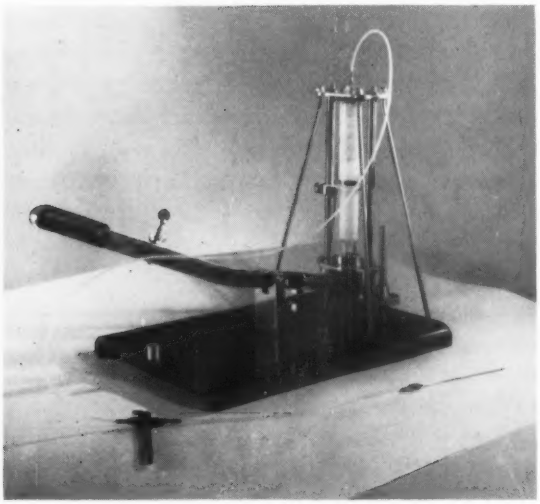

FIG. 1. Mechanical pressure injector.

In an effort to delineate the width and size of the regurgitant jet through the mitral valve in mitral insufficiency and to visualize more adequately the mitral valve leaflets, we have on several occasions employed the exaggerated right posterior oblique projection, but with caudal angulation of the central rays approximately 15 degrees. This projection resulted in partial overlap of the left ventricle on the left atrium, and did not improve our visualization of the mitral valve area. A 15 degree cephalad angulation was also tried, with the patient in left posterior oblique position. This produced some superimposition of the left atrium on the left ventricle.

FIG. 2. Basic equipment for cardiac ventriculography.

Further study may yield a more advantageous projection than our present direct vertical projection with the exaggerated right posterior oblique position.

Demonstration of aneurysms of the left ventricle involving the posterior ventricular wall, and lesions of the thoracic aorta, as well as visualization of the aortic valve appear to be best accomplished by the exaggerated right posterior oblique position.

A direct anteroposterior projection is recommended for the evaluation of tricuspid valvular insufficiency employing right ventriculography. This same projection seems preferable for the demonstration of left ventricular aneurysms involving the anterolateral wall of the left ventricle. Further experience may indicate the advisability of other projections.

With the patient in the desired position and with the heart and aorta properly centered over the filming area, a strip of adhesive tape holding a series of small lead numerals placed 1.0 cm. apart is then affixed in cephalocaudal axis to the skin of the patient's chest in the area of roentgen projection of the ventricle. A small lead marker is also placed at the site of skin puncture. With a palpable xiphoid cartilage, the lead marker is placed at the angle between the xiphoid and the left costochondral margin. When the xiphoid cartilage is not palpable, the marker is placed on the skin at the angle formed by the right and left costochondral cartilages. With the strip of lead numerals and the lead marker at the xiphoid area in place, a scout film is obtained. From this film (Fig. 3) the lead numeral most closely approximating the ultimate desired interventricular position of the needle tip is selected and used as a reference point, by ink-marking its location on the patient's skin. The ink-mark area serves as a guide for directing the degree of cephalad needle angulation.

TECHNIQUE OF VENTRICULAR PUNCTURE AND INJECTION

A diodrast skin test is a preliminary routine, even though we have been little

impressed with the value of this test.

A short run of electrocardiographic record is made. The syringe is loaded with the radiopaque substance. The tubing and needle assembly is then attached and the electromanometer connected through the three-way stopcock, with the tubing and needle assembly filled with physiological saline solution.

The skin puncture site in all of our series has been at the xiphoid area, as cardiac needle puncture at the surface of the heart closest to the xiphoid cartilage avoids major coronary arteries or larger coronary artery branches.

A procaine wheal is made at the puncture site, and the procaine local anesthesia extended through the chest wall. A small skin stab is made with a Number 15 knife blade.

For left cardiac ventriculography, the needle is aimed for a point half-way between the estimated position of the mitral valve and the apex of the left ventricle. With a normal-sized left ventricle, the needle is inserted toward the left, at a 10 degree angle to the sagittal plane of the body. With an enlarged left ventricle, the needle is inserted more closely to the sagittal plane. If the left ventricle is displaced by a large right ventricle, the needle is inserted at an approximate 20 degree angulation to the sagittal plane.

For right ventriculographic study, the needle is aimed at the estimated center of the right ventricle, with the needle, however, directed nearer to the sagittal axis of the patient.

Rapid needle passage is desirable. Continuous electrocardiographic recordings are obtained during the needle insertion. On intracardiac penetration of the needle, if the recorded pressure is lower than systemic blood pressure one must assume that *right* ventricular puncture has been effected. This can be verified by permitting a small amount of blood to enter the tubing. If the blood appears unoxygenated, one may be reasonably certain that the needle tip is in the right ventricle. When

Fig. 3. Preliminary film. Strip of lead numerals in cephalocaudal axis of patient projected over left ventricle. Lead marker at xiphoid area. The numeral most nearly approximating the desired ultimate position of the needle tip is selected as the aiming point for determination of degree of cephalad angulation of the needle.

the intent is *left cardiac ventriculography*, the needle is advanced farther through the ventricular septum, until left ventricular pressure is recorded. Figure 4 is a diagrammatic cross-section illustration of the relationships of the right ventricle, the interventricular septum and the left ventricle to the location of the needle effecting left ventricular puncture.

With penetration of the ventricle, the needle is slowly withdrawn until damping of the pressure tracing occurs. The needle is then readvanced 0.5 cm., or for such an additional distance as is required to obtain a good ventricular pressure recording. This maneuver, we feel, is of considerable importance in avoiding inadvertent medium infiltration of the septum or ventricular wall. With satisfactory and undamped ventricular pressures recorded, the stop-

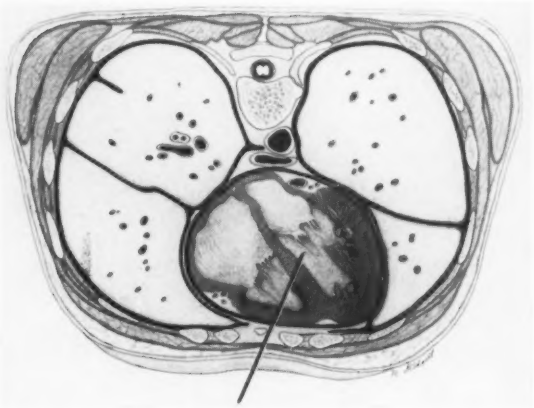

FIG. 4. Cross-sectional diagram of the right ventricle, interventricular septum and the left ventricle, and location of the needle effecting left ventricular puncture.

cock is turned to connect the pressure injector and the radiopaque medium is injected.

At the time of injection the patient is instructed to stop breathing. It is most important that the patient be cautioned against taking in a deep breath during the needle insertion, while the needle is in position, and at injection. The drawing of a deep breath has, on occasion, resulted in movement and displacement of the needle with resultant intramyocardial and, in one instance, intrapericardial injection. It is recommended that the total time for injection of the complete dosage of opaque medium not exceed two seconds. As soon as injection is completed, the needle is immediately withdrawn.

Continuous electrocardiographic record is maintained before, during and after the needle insertion and injection of the medium.

The filming procedure is started immediately prior to the beginning of the injection. The first of the series of twelve films thus serves as a "control" film for comparison with subsequent films showing ventricular opacification.

ANALYSIS OF CASES

This report presents our total experience with cardiac ventriculographies in humans. It comprises 77 studies in 60 patients. A tabulated résumé of these examinations is presented in Table 1. There were 22 males and 38 females. Age range was from sixteen to sixty-four years. Fifty-two patients were between the ages of twenty and fifty years; 2 were below twenty years of age and 6 were over fifty years old. In 17 examinations, two insertions of the needle were required to obtain what was considered at the time of the study to be a satisfactory ventricular puncture. In one examination, three needle passes were required.

More than one needle insertion was necessitated by either failure to obtain pressure tracing indicating ventricular puncture, or a pressure tracing showing a damped recording was obtained indicating that the needle tip was not clearly in the ventricular cavity but was, presumably, partially in myocardium and partially in the ventricular lumen.

In 2 examinations, failure to obtain the ventricular pressure record on the initial insertion of the needle was proved to be due to needle plugging by fragment of cartilage, probably the result of needle passage through the margin of the adjacent costal cartilage.

A satisfactory and undamped pressure tracing was not obtained in 4 examinations, and in these a film was immediately exposed before the needle was withdrawn. This "check" film revealed that the needle tip was too low in the ventricle, and the film aided in adjusting the direction for the second insertion of the needle.

An additional, though infrequent, reason for more than one puncture was the development of a severe cardiac arrhythmia, such as persistent ventricular tachycardia, occurring during myocardial puncture.

Of the total of 77 examinations, the objective was *left* cardiac ventriculography in 69. In 34 of these, needle passage was direct to the left ventricle without traversing the right ventricular cavity. In 32 left ventriculographies, the needle first traversed the

right ventricular cavity and a right ventricular pressure recording was obtained in these cases; the needle then passed through the septum to the left ventricular cavity.

Of the 69 examinations in which left ventriculography was the objective, "unintentional" right ventriculography was obtained 3 times, all these 3 occurring among the first 43 examinations. Such "unintentional" right ventriculography was attributable, in 2 of the 3 cases, to the presence of right ventricular hypertension resulting in recorded pressure values of magnitude simulating the expected left ventricular pressures. Experience has taught us that this error may be avoided, when there is uncertainty as to whether the needle tip is in the right or the left ventricle, by allowing a small amount of blood to reflux through the needle into the polyethylene tubing. Observation of the color of such blood, *i.e.*, whether it appears oxygenated or unoxygenated, will identify the chamber in which the needle tip is lodged.

The objective was *right* ventriculography in 8 examinations. There was one "unintentional" left ventricular opacification among these 8. This error is ascribed to an unreliable pressure monitoring.

SELECTION OF CASES

The purposes for which cardiac ventriculography was performed in our series of 77 examinations are as follows:

Evaluation of degree of mitral valvular insufficiency in patients with such clinical diagnosis	40
Demonstration of presence and degree, or absence, of mitral valvular insufficiency in patients with clinical diagnosis of mitral valvular disease	7
Evaluation of any residual mitral valve insufficiency following surgical procedure for this lesion	7
Evaluation of presence and/or degree of tricuspid valvular insufficiency	8
Demonstration of aortic valves in patients with clinical diagnosis of aortic valvular disease	5
Demonstration of suspected interventricular septal defect	3
Demonstration of left ventricular aneurysm	2
Evaluation of left ventricle following surgical procedure for left ventricular aneurysm	1
Demonstration of aortic aneurysm	2
Demonstration of coarctation of aorta	1
Evaluation of suspected and undiagnosed cardiovascular malformation	1

Figure 5 portrays a ventriculographic study in a patient with clinical diagnosis of mitral valvular insufficiency. Regurgitant opacification of the left atrium is clearly discernible. At surgery, a Grade 2 mitral insufficiency was found.

The progressive but not pronounced regurgitant opacification of a huge left atrium in a patient with surgically proved mitral insufficiency is demonstrated in Figure 6.

Figure 7 illustrates the application of cardiac ventriculography to the evaluation of surgical procedures for relief of mitral insufficiency. Preoperative ventriculography on this patient revealed a Grade 3 regurgitant opacification of the left atrium (grading on basis of 0 to 4); postoperative study revealed marked reduction of regurgitant opacification.

Regurgitant opacification of the right atrium, in a patient with clinical diagnosis of tricupsid insufficiency, is shown in Figure 8.

Figure 9 portrays the opacification of a left ventricular aneurysm, and the appearance of the ventricle after surgical resection of the aneurysm.

Figure 10 *A* illustrates the ventriculographic appearance of a normal aortic valve. Figure 10, *B* and *C* demonstrate the thickened appearance of aortic valve cusps in 2 patients with surgically proved aortic valvular disease. Figure 10 *D* depicts the aortic valve cusps of a patient with surgically proved aortic stenosis, mitral stenosis and a Grade 1 mitral insufficiency.

VENTRICULAR PRESSURE RECORDINGS

As indicated in the discussion of Technique of Ventricular Puncture and Injection, continuous electromanometric recording of intracardiac pressures, transmitted through the needle and tubing assembly, has been considered an essential component of our procedure. The pressure recordings form the basis for our determination

TABLE I

ANALYSIS OF 77 CARDIAC VENTRICULOGRAPHIES IN 60 PATIENTS

Patient	Age	Date	Purpose of Study	Amount Opaque (cc.)	Ventricular Puncture		Ventricular Pressure		Electrocardiographic Changes
					Objective	Needle Passage Course	Right	Left	Ventricular Puncture
1. J.L.	38	2/15/56	degree mitral insufficiency	30	L.V.	→R.V.	90/0	—	frequent ventricular prematures
2. R.R.	27	2/15/56	degree mitral insufficiency	30	L.V.	→L.V.	unavailable	unavailable	frequent ventricular prematures; ventricular tachycardia 6-beat
3. "	27	4/23/56	postoperative evaluation	30	L.V.	→R.V.→L.V.	40–60/0	105–115/0	3 ventricular prematures
4. K.G.	64	2/17/56	evaluate aortic aneurysms	30	L.V.	→R.V.→L.V.	unavailable	unavailable	occasional ventricular prematures; ventricular tachycardia 8-beat
5. J.M.	25	2/20/56	degree mitral insufficiency	30	L.V.	→R.V.→L.V.	32/0	100/10	coupling 2-beat; aberrant conduction with ventricular prematures and fused beats
6. M.M.	28	2/20/56	evaluate aortic valve and subvalvular area	30	L.V.	→L.V.	—	120/0	ST segment elevation
7. G.N.	39	2/20/56	degree mitral insufficiency	30	L.V.	→L.V.	—	140/35	frequent ventricular prematures with coupling
8. E.K.	31	2/29/56	degree mitral insufficiency	30	L.V.	→L.V.	—	135/5	none
9. T.G.	51	3/2/56	demonstrate suspected interventricular septal defect	30	L.V.	1) tubing disconnected 2) →L.V.	—	90–110/5	frequent ventricular prematures; ventricular tachycardia 11-beat; atrio-ventricular dissociation
10. A.D.	20	3/8/56	degree mitral insufficiency	30	L.V.	1) →R.V. 2) →R.V.→L.V.	30/0	100–110/10	ventricular tachycardia 11-beat
11. S.O.	44	3/13/56	degree mitral insufficiency	30	L.V.	→L.V.	—	140/5	frequent ventricular prematures; ventricular tachycardia 6-beat
12. "	44	3/19/56	degree mitral insufficiency (repeat study)	30	L.V.	→L.V.	—	115/0–10	none
13. F.B.	39	3/19/56	evaluate for any mitral insufficiency	40	L.V.	1) cartilage plugging 2) →R.V.→L.V.	55/0	150/5	frequent ventricular prematures; right bundle branch block; delayed intraventricular conduction
14. L.K.	33	3/19/56	degree mitral insufficiency	25	L.V.	→R.V.→L.V.	unreliable	150–160/0–5	ventricular tachycardia 5-beat
15. L.B.	51	3/27/56	degree mitral insufficiency	30	L.V.	→L.V.	—	120/0	frequent ventricular prematures; ventricular tachycardia 14-beat; occasional coupling; bradycardia
16. F.S.	49	3/27/56	degree mitral insufficiency	30	L.V.	→L.V.	—	125/20	ventricular tachycardia 3, 5-beat
17. "	49	4/19/56	postoperative evaluation	35	L.V.	→L.V.	—	110/0–5	2 ventricular prematures
18. M.S.	21	4/2/56	degree mitral insufficiency	40	L.V.	→L.V.	—	135/10	2 ventricular prematures
19. "	21	4/19/56	postoperative evaluation	40	L.V.	→R.V.→L.V.	60/0	135/10	1 ventricular premature
20. S.C.	42	4/3/56	evaluate left ventricular aneurysm	40	L.V.	1) cartilage plugging 2) →L.V.	—	100–105/0	2 nodal beats; 1 ventricular premature; ventricular tachycardia 7-beat; nodal escape; wandering pacemaker
21. "	42	4/25/56	postoperative evaluation	40	L.V.	→R.V.→L.V.	28/0	100/0	frequent ventricular prematures
22. L.C.	57	4/5/56	demonstrate suspected left ventricular aneurysm	35	L.V.	→R.V.	100/0	—	coupling to bigeminal rhythm
23. V.V.	49	4/5/56	degree mitral insufficiency	40	L.V.	→R.V.→L.V.	30/0	80/0	occasional ventricular prematures
24. M.H.	24	4/6/56	demonstrate suspected coarctation of aorta	30	L.V.	→L.V.	—	140/0	ventricular tachycardia 7, 3, 10-beat; some fused beats
25. M.H.	42	4/6/56	degree mitral insufficiency	30	L.V.	→L.V.	—	125/5	occasional ventricular prematures; ventricular tachycardia 6-beat
26. "	42	4/26/56	postoperative evaluation	40	L.V.	1) →R.V. 2) →R.V.→L.V.	30/5	120/0	occasional ventricular prematures
27. E.F.	27	4/10/56	degree mitral insufficiency	40	L.V.	→R.V.→L.V.	40/0	90–115/10	frequent ventricular prematures; ventricular tachycardia 4-beat
28. "	27	5/2/26	postoperative evaluation	40	L.V.	→R.V.→L.V.	40/10	90–115/10	ventricular flutter 8-beat
29. A.P.	43	4/10/56	evaluate for any mitral insufficiency	30	L.V.	→L.V.	—	120/0	occasional ventricular prematures
30. M.B.	28	4/11/56	degree mitral insufficiency	40	L.V.	→R.V.→L.V.	65–70/5–10	125–150/10–15	frequent ventricular prematures; ventricular tachycardia 7-beat

AI = Aortic valvular insufficiency
AS = Aortic valvular stenosis
CHD = Congenital heart disease
IVSD = Interventricular septal defect
L.V. = Left ventricle
MI = Mitral valvular insufficiency
MS = Mitral valvular stenosis
R.A. = Right atrium
R.V. = Right ventricle
TI = Tricuspid valvular insufficiency

Electrocardiographic Changes		Diagnosis				Examination Quality		Complications
Diodrast Injection	Post-Injection	Pre-Examination	Examination	Final	Verification	Grading	Explanation	
none	frequent ventricular premature	MI	none	MI major MS minor	clinical	failure	unintentional R.V. injection; high R.V. pressure	none
none	none	MI	MI+++	MI+++	surgical	good		none
2 ventricular premature	ventricular tachycardia 9-beat	—	MI+	—	—	good		none
none	none	aortic aneurysms	aortic aneurysms	aortic aneurysms	surgical	excellent		none
2 ventricular premature; bradycardia 4 seconds	tachycardia	MI; AS(?)	MI+++; aortic valve not thickened	MI+++	surgical	excellent		none
2 ventricular premature; ST segment elevation (short duration)	none	sub-aortic stenosis	aortic valve thickened	aortic stenosis (congenital)	clinical	poor	needle tip too high in outflow tract	none
ventricular tachycardia 9-beat; short ventricular flutter	frequent coupling	MI major AS minor	none	MI major AS minor	surgical	failure	intramyocardial injection (partial)	intramyocardial injection (moderate); mild substernal pain; mild shock (short duration); recovery
none	ventricular tachycardia 4-beat	MI	MI+++	MI+++	surgical (autopsy)	good		none
atrioventricular dissociation	ST segment elevation	IVSD (?); AI (?)	none	IVSD; AI	surgical	failure	intramyocardial/intrapericardial injection (partial)	intramyocardial/intrapericardial injection (moderate); substernal pain; shock—24 hr. duration; recovery
1 ventricular premature; bradycardia 3 seconds	tachycardia	MI; AI	MI++; aortic valve thickened (?)	MI; AI	clinical	fair		none
frequent ventricular prematures	none	MI; MS	none	—	—	failure	intramyocardial/intrapericardial injection (entire); no ventricular opacification	intramyocardial/intrapericardial injection (total); slight pain, no shock
ventricular tachycardia 4-beat	none	MI; MS	none	MI++	surgical	failure	needle tip too high—in outflow tract	none
changing aberrant conduction through bundle	atrioventricular dissociation	MS major; MI, AS, AI—all minor	no MI; aortic valve thickened	MS; AS; TI	surgical	poor	intramyocardial injection (partial)	intramyocardial injection (moderate); slight pain; no shock
ventricular tachycardia 3-beat	none	MI; AI major MS; AS minor	no MI; aortic valve thickened	MS; AI major MI; AS minor	clinical	fair		none
3 ventricular prematures	none	MI; MS	MI++	MI	clinical	good		none
none	none	MI major MS minor	MI++	MI+++	surgical	fair		none
2 ventricular prematures; bradycardia 6 seconds	none	—	MI++	—	—	good		none
none	none	MI	MI+++	MI+++; MS	surgical	excellent		none
2 ventricular prematures; bradycardia 4 seconds	tachycardia	—	MI+; aortic valve thickened (?)	—	—	excellent		none
bradycardia 6 seconds	tachycardia	left ventricular aneurysm	left ventricular aneurysm	left ventricular aneurysm	surgical	excellent		none
2 ventricular prematures	none	—	aneurysm resected	—	—	excellent		none
supraventricular tachycardia; aberrant conduction; right bundle branch block	supraventricular tachycardia; aberrant conduction; right bundle branch block	left ventricular aneurysm (?)	none	left ventricular aneurysm	surgical	failure	unintentional R.V. injection; high R.V. pressure; intramyocardial injection (partial)	intramyocardial injection (moderate); palpitation; no pain; no shock
ventricular tachycardia 4-beat; 4.6 second ventricular asystole	tachycardia	MI	MI+++	MI++ MS minor	surgical	excellent		none
1 ventricular premature	none	coarctation of aorta	coarctation of aorta	coarctation of aorta	surgical	good		none
1 ventricular premature	occasional coupling	MI	MI++	MI+++	surgical	good		none
5 ventricular prematures; brachycardia 3 seconds	tachycardia	—	MI+++	—	—	good		none
ventricular tachycardia 4-beat	none	MI major MS minor	MI++++	MI++++ MS minor	surgical	excellent		none
ventricular tachycardia 7-beat; bradycardia 2 seconds	none	—	no MI	—	—	excellent		none
record unreliable	record unreliable	MS	no MI	MS pure	surgical	excellent		none
bradycardia 3 seconds	tachycardia	MI	MI++++	MI+++	surgical	excellent		none

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Patient	Age	Date	Purpose of Study	Amount Opaque (cc.)	Ventricular Puncture		Ventricular Pressure		Electrocardiographic Changes
					Objective	Needle Passage Course	Right	Left	Ventricular Puncture
31. B.S.	26	4/11/56	evaluate for any tricuspid insufficiency	20	R.V.	→R.V. →L.V.	unreliable	unreliable	occasional coupling; ventricular tachycardia 3, 5, 3, 6-beat
32. "	26	4/12/56	evaluate for any tricuspid insufficiency (repeat)	20	R.V.	→R.V.	15–20/0	—	ventricular tachycardia 5-beat
33. M.R.	45	4/12/56	degree mitral insufficiency	40	L.V.	→L.V.	—	115/0	1 ventricular premature
34. R.C.	31	4/13/56	degree mitral insufficiency	30	L.V.	→L.V.	—	110–125/0	frequent ventricular prematures
35. "	31	5/ 3/56	postoperative evaluation	40	L.V.	→L.V.	—	110–125/0	2 ventricular prematures
36. J.A.	21	4/13/56	degree mitral insufficiency	40	L.V.	1) →L.V. →myocardium 2) →R.V. →L.V.	35/0	115/0	coupling 4-beat; ventricular tachycardia 4, 9-beat
37. "	21	5/ 2/56	postoperative evaluation	40	L.V.	→R.V. →L.V.	25–35/0	120/0	ventricular tachycardia 5, 8-beat
38. C.R.	16	4/16/56	demonstrate suspected interventricular septal defect	35	L.V.	→L.V.	—	150–170/10	none
39. A.F.	32	4/20/56	degree tricuspid insufficiency	40	R.V.	→R.A. →R.V.	60–75/0–5	—	bigeminal rhythm; ventricular tachycardia 3-beat
40. "	32	4/23/56	degree tricuspid insufficiency (repeat)	60	R.V.	→R.V.	60–75/0–5	—	occasional ventricular prematures
41. "	32	4/26/56	degree mitral insufficiency	40	L.V.	→R.V. →L.V.	65/5	120–130/5–15	occasional ventricular prematures; ventricular tachycardia 5-beat
42. R.H.	34	4/24/56	evaluate for any mitral insufficiency	10	L.V.	→R.V. →L.V.	20–30/0	120/0	2 ventricular prematures
43. "	34	4/25/56	evaluate for any mitral insufficiency (repeat)	40	L.V.	1) →L.V. →myocardium 2) →R.V. →L.V. →myocardium →R.V. →septum	45/0	115/0	occasional ventricular prematures
44. T.M.	49	4/27/56	evaluate aortic valve	40	L.V.	→L.V.	—	160/5	frequent ventricular prematures; ventricular tachycardia 4,4-beat
45. M.D.	38	4/27/56	degree tricuspid insufficiency	50	R.V.	→R.V.	40–50/0	—	frequent ventricular prematures; ventricular tachycardia 3-beat
46. E.B.	50	4/30/56	degree tricuspid insufficiency	40	R.V.	1) too low—myocardium 2) too low—myocardium 3) →R.V.	40–50/0	—	occasional coupling; ventricular tachycardia 3-beat
47. "	50	5/ 1/56	evaluate for any mitral insufficiency	40	L.V.	→R.V. →L.V.	40–50/0	60–90/0	occasional ventricular prematures; ventricular tachycardia 5-beat
48. I.S.	21	4/30/56	degree mitral insufficiency	40	L.V.	→R.V. →L.V.	50/10	115–130/0–10	2 ventricular prematures; ventricular tachycardia 4-beat
49. "	21	5/ 1/56	degree tricuspid insufficiency	50	R.V.	1) too low—myocardium 2) →R.V. →L.V. →R.V.	50/10	120/20	ventricular tachycardia 4, 4, 3, 8, 3-beat
50. M.W.	24	5/ 1/56	degree mitral insufficiency	40	L.V.	→R.V. →L.V.	20/0	95–115/5	ventricular tachycardia 4-beat
51. C.I.	31	5/14/56	degree mitral insufficiency	40	L.V.	→L.V.	—	135/0	ventricular tachycardia 3-beat
52. C.H.	28	5/21/56	degree mitral insufficiency	37	L.V.	→L.V.	—	135/0–5	occasional ventricular prematures
53. L.D.	31	5/23/56	degree mitral insufficiency	45	L.V.	1) too low—myocardium 2) →L.V.	—	130/0	frequent ventricular prematures; ventricular tachycardia 4, 3, 5, 6, 14-beat
54. M.B.	27	5/29/56	degree mitral insufficiency	40	L.V.	→L.V.	—	135/0	frequent ventricular prematures; ventricular tachycardia 11, 6-beat
55. F.D.	23	5/29/56	degree mitral insufficiency	10	L.V.	→L.V.	—	110/10	occasional ventricular prematures; ventricular tachycardia 5-beat; nodal escape 1-beat
56. M.W.	49	6/ 5/56	degree tricuspid insufficiency	40	R.V.	→R.V.	80/10	—	frequent coupling; ventricular tachycardia 3-beat
57. "	49	6/14/56	degree mitral insufficiency	45	L.V.	→R.V. →L.V.	80–100/10	160/10	ventricular tachycardia 3, 3, 4, 4-beat
58. C.C.	32	6/ 5/56	degree mitral insufficiency	40	L.V.	→R.V. →L.V.	25/0–10	100/0	frequent ventricular prematures
59. L.K.	49	6/ 5/56	degree mitral insufficiency	40	L.V.	1) →L.V. →myocardium 2) →R.V. →L.V.	30/0–5	90–95/0–5	occasional ventricular prematures; ventricular tachycardia 5-beat; bradycardia
60. W.T.	45	6/11/56	degree mitral insufficiency	10	L.V.	1) →R.V. →myocardium 2) →L.V.	—	150/0–10	occasional ventricular prematures; ventricular tachycardia 3-beat
61. J.V.	41	6/12/56	evaluate for suspected congenital heart disease	40	L.V.	1) →L.V. →myocardium 2) →R.V. →L.V.	30/0	125/0	1 ventricular premature; ventricular tachycardia 5-beat
62. H.B.	47	6/18/56	evaluate aortic valve	40	L.V.	→L.V.	—	165/0–10	none

Electrocardiographic Changes		Diagnosis				Examination Quality		Complications
Diodrast Injection	Post-Injection	Pre-Examination	Examination	Final	Verification	Grading	Explanation	
ventricular tachycardia 6-beat; bradycardia 3 seconds	tachycardia	CHD with TI	none	—	—	failure	unintentional L.V. injection	none
none	none	CHD with TI	none	CHD; huge right atrium	surgical	failure	needle tip too high in outflow tract	none
coupling 2-beat; bradycardia 3 seconds	ST segment depression and T inversion; tachycardia	MI; MS	MI++	MI+++	surgical (autopsy)	excellent		none
2 ventricular prematures	none	MI; MS	MI++++; aortic valve thickened	MI++	surgical	excellent		none
3 ventricular prematures; bradycardia 4 seconds	tachycardia	—	no MI; aortic valve thickened	—	—	excellent		none
2nd degree A-V block; bradycardia 2 seconds	none	MI	MI+++	MI+++ MS minor	surgical	excellent		none
ventricular tachycardia 5-beat; nodal rhythm	none	—	no MI	—	—	excellent		none
few ventricular prematures; sinus arrest 2 seconds; nodal rhythm; bradycardia 5 seconds	tachycardia	IVSD (?)	normal	IVSD (?)	clinical	good		anaphylactic shock (severe)—10 min. post-injection; continued 10 hours; recovery
ventricular tachycardia 6-beat	none	TI; MI	none	—	—	failure	improper film positioning of patient	none
ventricular tachycardia 5-beat	none	TI; MI	No TI	—	—	excellent		none
none	none	MI	MI++	MI	surgical (autopsy)	fair		none
none	none	MI; MS	MI++	—	—	poor	syringe breakage; only 10 cc. injected	none
ventricular asystole 6.5 seconds; complete heart block—rate 37/min.	complete heart block	MI; MS	none	MI	clinical	failure	unintentional R.V. injection; interventricular septal injection (partial)	septal injection (partial-slight); no pain; no shock; immediate complete heart block (persists to present, 4 months)
ventricular tachycardia 3, 4-beat; bradycardia 4 seconds	tachycardia	AS	aortic valve thickened; no MI	AS	surgical	excellent		none
ventricular tachycardia 6-beat; coupling 3-beat	none	TI; MS	TI++++	TI major MS minor	clinical	excellent		intramyocardial injection (minimal); no pain; no shock
ventricular tachycardia 4-beat; coupling 2-beat	none	TI; MS; MI	TI++++	—	—	excellent		none
ventricular tachycardia 7-beat; occasional coupling	none	TI; MS; MI	no MI	TI; MI	clinical	good		intramyocardial injection (minimal); no pain; no shock
4 ventricular prematures	none	MI; TI	MI++++	—	—	excellent		none
ventricular tachycardia 3,5-beat; occasional coupling	tachycardia	MI; TI	TI++++	MI++++ TI	surgical	excellent		none
ventricular tachycardia 3, 3-beat; bradycardia 4 seconds	tachycardia	MI major MS minor	MI+++	MI+++	surgical	good		none
ventricular tachycardia 6-beat; bradycardia 3 seconds	tachycardia	MI; MS	MI+++	MI	clinical	excellent		none
2 ventricular prematures; bradycardia 4 seconds	none	MI	MI++	MI	clinical	poor	patient of unusually large habitus	none
ventricular tachycardia 5-beat	none	MI	MI+++	MI++++	surgical (autopsy)	good		none
sinus arrest 2 seconds; nodal escape 1-beat; bradycardia 3 seconds	tachycardia	MI	none	MI	clinical	failure	seriographic failure—no films	none
2 ventricular prematures	none	MI	none	MI	clinical	failure	syringe breakage; 10 cc. injection	none
ventricular tachycardia 5, 5-beat	none	TI; MI major MS minor	TI+++	—	—	excellent		chills, fever—1 hr. post-procedure; pyrogen reaction (?); recovery
occasional ventricular prematures	tachycardia	TI; MI major MS minor	MI++	MI; TI	clinical	good		none
2 ventricular prematures; bradycardia 3 seconds	tachycardia	MI	MI+++	MI; MS	clinical	excellent		none
1 ventricular premature; bradycardia 12 seconds	none	MI; MS	MI+++	MI+++	surgical (autopsy)	excellent		none
1 ventricular premature	none	MI	no MI	no valvular disease	clinical	fair	tubing disconnected; 10 cc. injection	none
1 ventricular premature; bradycardia 4 seconds	tachycardia	CHD (?)	normal	no significant lesion	clinical	good		none
ventricular tachycardia 3-beat; bradycardia 4 seconds	tachycardia	AS	aortic valve thickened; no MI	AS	clinical	good		none

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ANALYSIS OF 77 CARDIAC VENTRICULOGRAPHIES IN 60 PATIENTS

Patient	Age	Date	Purpose of Study	Amount Opaque (cc.)	Ventricular Puncture		Ventricular Pressure		Electrocardiographic Changes
					Objective	Needle Passage Course	Right	Left	Ventricular Puncture
63. R.H.	40	6/18/56	degree mitral insufficiency	45	L.V.	→L.V.	—	110–125/0–10	1 ventricular premature; ventricular tachycardia 3, 4, 4, 6-beat
64. W.P.	56	6/18/56	degree mitral insufficiency	45	L.V.	→L.V.	—	110–140/0	none
65. G.S.	36	6/21/56	degree mitral insufficiency	40	L.V.	→L.V.	—	130/0–20	occasional ventricular premature; ventricular tachycardia 3-beat
66. S.B.	39	6/27/56	evaluate aortic valve	25	L.V.	→L.V.	—	200/10	none
67. H.D.	39	7/2/56	evaluate for any mitral insufficiency	40	L.V.	→L.V.	—	110–140/0–5	none
68. J.N.	24	7/2/56	degree mitral insufficiency	40	L.V.	1) →R.V. 2) →R.V.→L.V.	40/0–5	115/0	occasional ventricular premature; occasional coupling; ventricular tachycardia 3, 5, 6-beat
69. G.D.	40	7/3/56	evaluate for any mitral insufficiency	40	L.V.	1) →L.V.—myocardium 2) →L.V.	—	110/0–10	frequent ventricular premature; bigeminal rhythm; ventricular tachycardia 3, 3, 5, 5, 9-beat
70. M.W.	28	7/6/56	degree mitral insufficiency	40	L.V.	→R.V.→L.V.	60/0–5	140/10	ventricular tachycardia 3, 6-beat
71. B.L.	56	7/6/56	degree mitral insufficiency	40	L.V.	→R.V.→L.V.	65/0	95/0	occasional ventricular premature
72. "	56	7/10/56	degree mitral insufficiency (repeat)	40	L.V.	1) →R.V.→L.V.—myocardium 2) →R.V.→L.V.	60/0–5	95–100/0	2 ventricular premature; ventricular tachycardia 3, 7, 7-beat
73. G.G.	35	7/9/56	degree mitral insufficiency	40	L.V.	1) →R.V.→L.V.—myocardium 2) →R.V.→L.V.	35/5	160/0–5	occasional ventricular premature; ventricular tachycardia 5, 3-beat; bradycardia
74. E.B.	28	7/10/56	degree mitral insufficiency	40	L.V.	→R.V.→L.V.	45/0–5	100–105/5–10	ventricular tachycardia 3, 6-beat
75. F.A.	44	7/20/56	evaluate aortic valve	40	L.V.	1) too low—myocardium 2) →L.V.	—	220/0	none
76. N.S.	18	7/26/55	demonstrate suspected interventricular septal defect	40	L.V.	→R.V.→L.V.	200/0	100/0–5	frequent ventricular premature
77. J.N.	43	8/7/56	evaluate aortic aneurysm	50	L.V.	→L.V.	—	125/0	ventricular tachycardia 3-beat

of the entrance of the needle tip into the ventricular cavity.

Of the 69 examinations in which *left* cardiac ventriculography was the objective, the *right* ventricle was traversed in 32, with pressure recordings being obtained for *both* ventricles. Figure 11 demonstrates the pressure recording as the needle tip passes from the right ventricle, through the septum, into the left ventricle. The pressure record with regard to the right ventricle in one of the 32 examinations was, upon analysis, considered unreliable, and the pressure records for one other of these 32 examinations are unavailable.

Of the 30 ventriculographies in which both right and left ventricular pressure records are available and reliable, only one showed a right ventricular systolic pressure equal to or exceeding that of the left ventricle. In this one case, the right ventricular systolic pressure was 200 mm. Hg, while the left ventricular systolic pressure was but 100 mm. Hg. At subsequent surgery, this pressure differential was ascribed to a severe, pure pulmonic stenosis.

As previously mentioned, the needle passage was direct to the left ventricle in 34 instances of left ventricular puncture. Figure 12 demonstrates the pressure and electrocardiographic tracings of such direct left ventricular puncture.

The systolic pressure in the left ventricle is normally of the same order of magnitude as the systemic systolic pressure. In patients with dynamic aortic stenosis, a gradient exists between the left ventricular systolic pressure and the systemic systolic pressure. In 10 patients an additional intent of the procedure was to obtain data regarding the presence or absence of such a gradient, and the systemic systolic pressure was recorded through an in-dwelling Cournand needle inserted in a systemic artery.

Right ventricular pressures were recorded in all of the 8 examinations in which right ventriculography was the objective.

Electrocardiographic Changes		Diagnosis				Examination Quality		Complications
Diodrast Injection	Post-Injection	Pre-Examination	Examination	Final	Verification	Grading	Explanation	
ventricular tachycardia	tachycardia	MI; MS	MI++	MI; MS	clinical	excellent		none
6-beat occasional ventricular prematures with fused beats	occasional ventricular prematures with fused beats	MS major MI minor	MI+	MI	clinical	good		none
none	none	MI major MS minor	MI++	MI major MS minor	clinical	fair		none
none	none	AS	aortic valve thickened; no MI	AS (pure)	surgical	fair	syringe breakage; 25 cc. injection	none
none	none	MS major MI minor	no MI	MS	clinical	good		none
bradycardia 4 seconds	none	MI	none	MS major MI; TI minor	surgical	failure	needle tip too high in outflow tract	none
ventricular tachycardia 7-beat	tachycardia	MS	no MI	MS	clinical	excellent		none
ventricular tachycardia 4-beat; bigeminal rhythm – 8 seconds	none	MI major MS minor	MI+	MI+++	surgical	good		none
occasional ventricular prematures	none	MI	MI++++	—	—	excellent	needle tip at mitral valve (?)	none
2 ventricular prematures	none	MI	MI++++	MI	clinical	excellent	re-check examination	none
none	none	MI; AS major AI minor	MI+; aortic valve thickened	AS; MS; MI+	surgical	good		none
1 ventricular premature; bradycardia 5 seconds	bradycardia	MI; MS	MI+++	MI; MS	clinical	good		none
4 ventricular prematures	none	AS	aortic valve thickened; no MI	AS	surgical	good		none
none	none	IVSD (?)	no IVSD	pulmonary stenosis (pure)	surgical	excellent		none
bradycardia 4 seconds	none	aortic aneurysm	aortic aneurysm	aortic aneurysm	surgical	excellent		none

Pressure records are available and reliable for 7 of these examinations.

The procedure of slight withdrawal of the needle, after initial ventricular puncture, until damping of pressure waves is evident and then slight re-advancement of the needle as a part of the needle puncture technique has been previously mentioned. The typical damping of ventricular pressure waves produced by this maneuver is illustrated in Figure 13.

The pressure tracing provides important and useful information as to whether the needle tip is clearly within the ventricular cavity. A partially damped wave is an indication that the needle tip in some measure engages the ventricular wall or a papillary muscle, and that there is need for readjustment of the needle. Figure 14 depicts a pressure tracing showing dampening of the left ventricular pressure waves. The tracing indicates that the needle tip is in the left ventricular chamber during the first portion of ventricular systole. During the latter portion of systole and the succeeding diastole, a damped curve is obtained, indicating that at this portion of the ventricular movement the needle tip is at least partially in the ventricular wall.

ELECTROCARDIOGRAPHIC OBSERVATIONS

The intracardiac insertion of a needle can, and rather regularly does, produce some disturbance of cardiac rhythm. On occasion, the needle insertion may also produce conduction aberrations.

It is also well established that the passage of a high concentration of radiopaque medium, such as 70 per cent diodrast, through the heart can produce temporary electrocardiographic changes.

In the performance of cardiac ventriculography, we have evidence that the impingement upon the ventricular endocardium of the jet of radiopaque medium issuing from the needle can produce electrocardiographic disturbances.

Our observations regarding electrocardi-

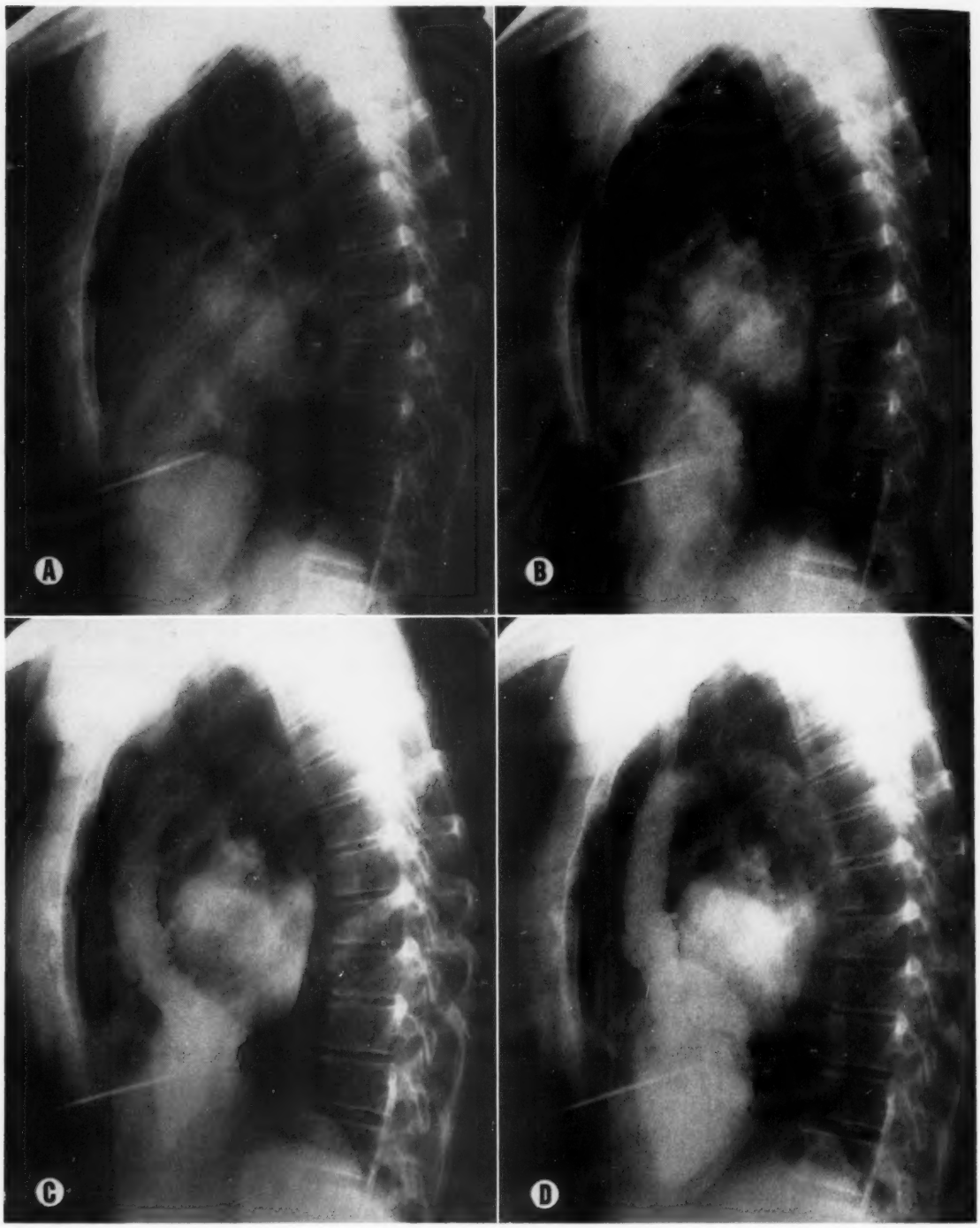

FIG. 5. Case V. V. Cardiac ventriculography—left. Mitral Valvular Insufficiency. (*A*) First film of twelve-film series, obtained $\frac{1}{2}$ second before start of injection. This film serves as scout control. (*B*) Beginning opacification of left ventricle. (*C*) Film at one second (20 cc. diodrast injected). Marked regurgitant opacification of left atrium. (*D*) Film at 2 seconds (completion of injection 40 cc. diodrast). Ventricle in diastole. Left atrium well opacified. Regurgitation estimated at Grade 3. Note visualization of mitral valve leaflets, aortic valve cusps and coronary vessels. Surgically proved mitral insufficiency, Grade 2.

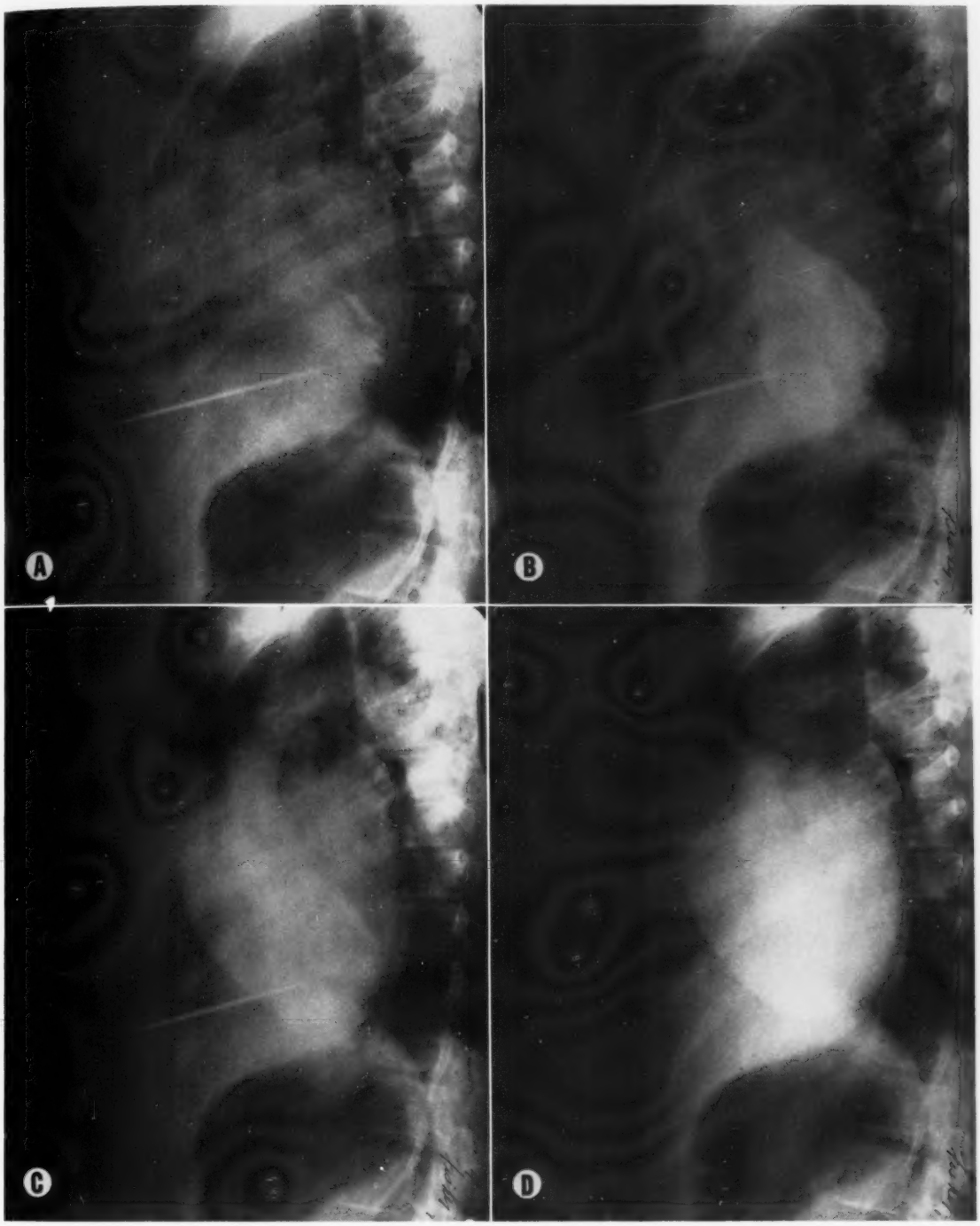

Fig. 6. Case E. F. Cardiac ventriculography—Left. Mitral valvular insufficiency. (*A*) Beginning of injection. (*B*) One-half second later. Opacification of left ventricle. (*C*) Film at approximately two seconds (completion of injection 40 cc. diodrast). Regurgitant opacification of huge left atrium. (*D*) Film at three seconds. Further opacification of huge left atrium. Regurgitation estimated as Grade 4; proved as Grade 4 at surgery.

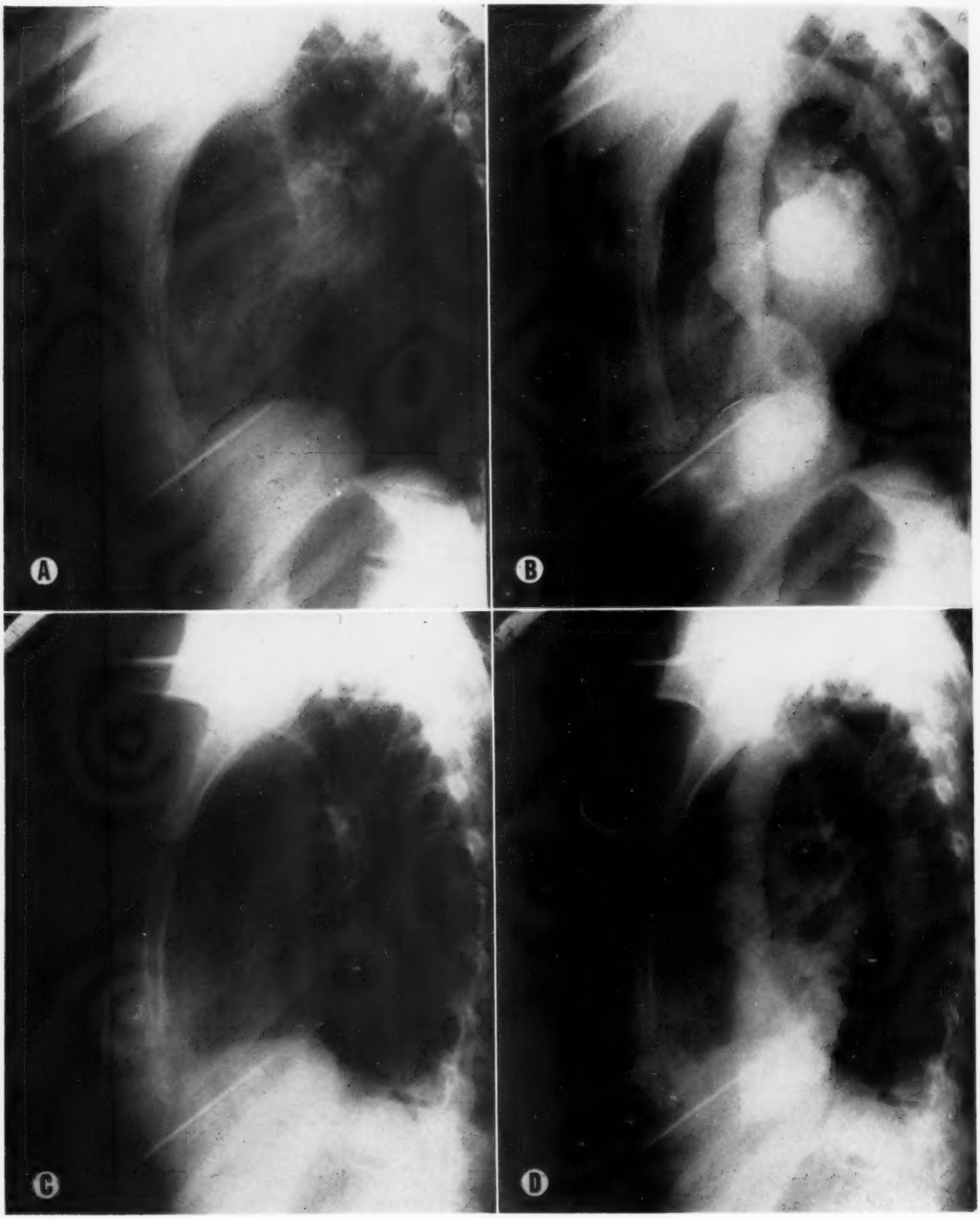

FIG. 7. Case M. S. Cardiac ventriculography—Left. Mitral valvular insufficiency. (*A*) Preoperative study. First film (immediately before injection). (*B*) Film at one and one-half seconds (near completion of injection 40 cc. diodrast). Marked regurgitant opacification of left atrium, estimated at Grade 3, and surgically proved as Grade 3. (*C*) Postoperative study 14 days following cross plication posterior commissure of mitral valve. First film (immediately before injection). (*D*) Film at one and one-half seconds (near completion of injection 40 cc. diodrast).Marked reduction of regurgitant opacification of left atrium (estimated Grade 1 or less).

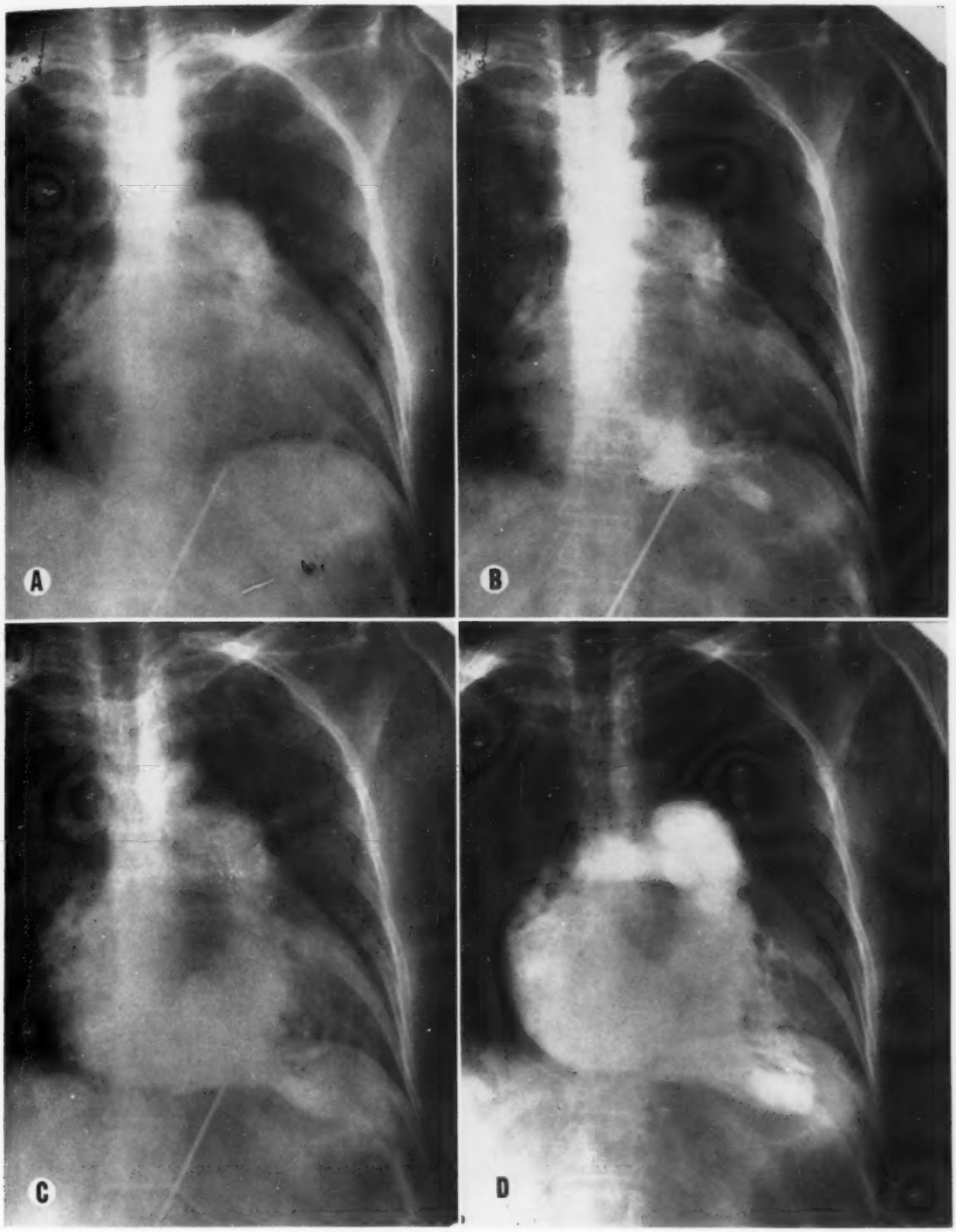

Fig. 8. Case E. B. Cardiac ventriculography—right. Tricuspid valvular insufficiency. (*A*) First film, immediately before injection. (*B*) One-half second later. Beginning of injection. (*C*) Film at approximately one second after start of injection. Partial opacification of right ventricle and beginning regurgitant opacification of right atrium. (*D*) Film at two and one-half seconds. Approximately one-half second after completion of injection 50 cc. diodrast. Excellent opacification of right ventricle and regurgitant opacification of right atrium. No surgical confirmation but clinical evidence of tricuspid insufficiency.

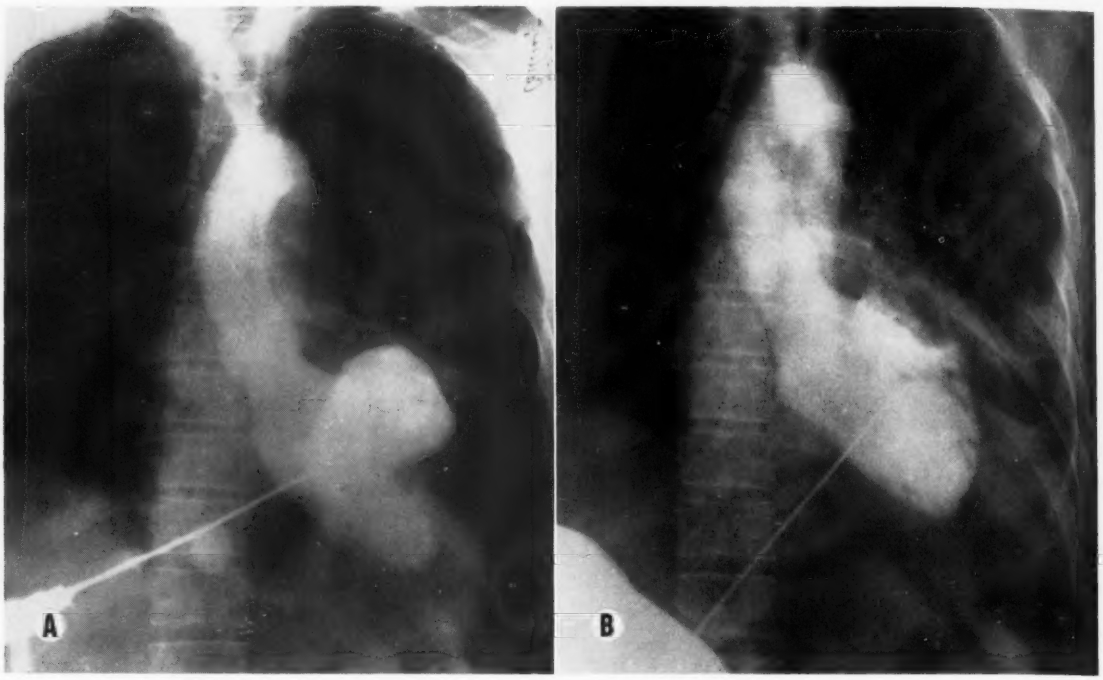

FIG. 9. Case S. C. Cardiac ventriculography—left. Aneurysm of left ventricle. (*A*) Preoperative study. Film at approximately one and one-half seconds (near completion of injection 40 cc. diodrast). Excellent opacification of left ventricular aneurysm. (*B*) Postoperative study twenty days after surgery. Film at two seconds (completion of injection 40 cc. diodrast) demonstrates resection of aneurysm.

ographic changes incident to cardiac ventriculography have been almost entirely confined to electrocardiograms obtained by a standard lead II. Obviously, such a record is not a complete cardiographic analysis, but the ventriculographic procedure as performed by us does not permit of simultaneous multiple lead and chest lead electrocardiographic recordings.

Our most common electrocardiographic observation during cardiac ventriculography is as follows: As the needle tip contacts the heart muscle, several ventricular premature beats occur, or a short run of ventricular tachycardia is observed. With advancement of the needle through the myocardium, there are frequently a few additional ventricular prematures or, on occasion, a short run of ventricular tachycardia. If the needle traverses the septum, there are frequently additional ventricular premature beats or a short run of ventricular tachycardia. Upon entry of the needle into the ventricular chamber, and with no further needle manipulation, the arrhythmia usually disappears. At injection of the radiopaque medium, a 4- to 9-beat run of ventricular tachycardia is a not infrequent finding. Upon withdrawal of the needle, the pre-examination electrocardiographic pattern is usually resumed.

Of the total series of 77 cardiac ventriculographies, the entire procedure was accomplished without evidence of even the slightest arrhythmia or any significant change in heart rate in only two examinations.

We have observed that *slow* intracardiac insertion of the needle produced more frequent ventricular premature beats and ventricular tachycardia of longer duration than did rapid needle advancement (Fig. 12).

Inadvertent intramyocardial or intrapericardial injection of diodrast can produce more profound electrocardiographic disturbances. There were 8 cases in which such inadvertent injection was made, and these will be discussed later in this paper.

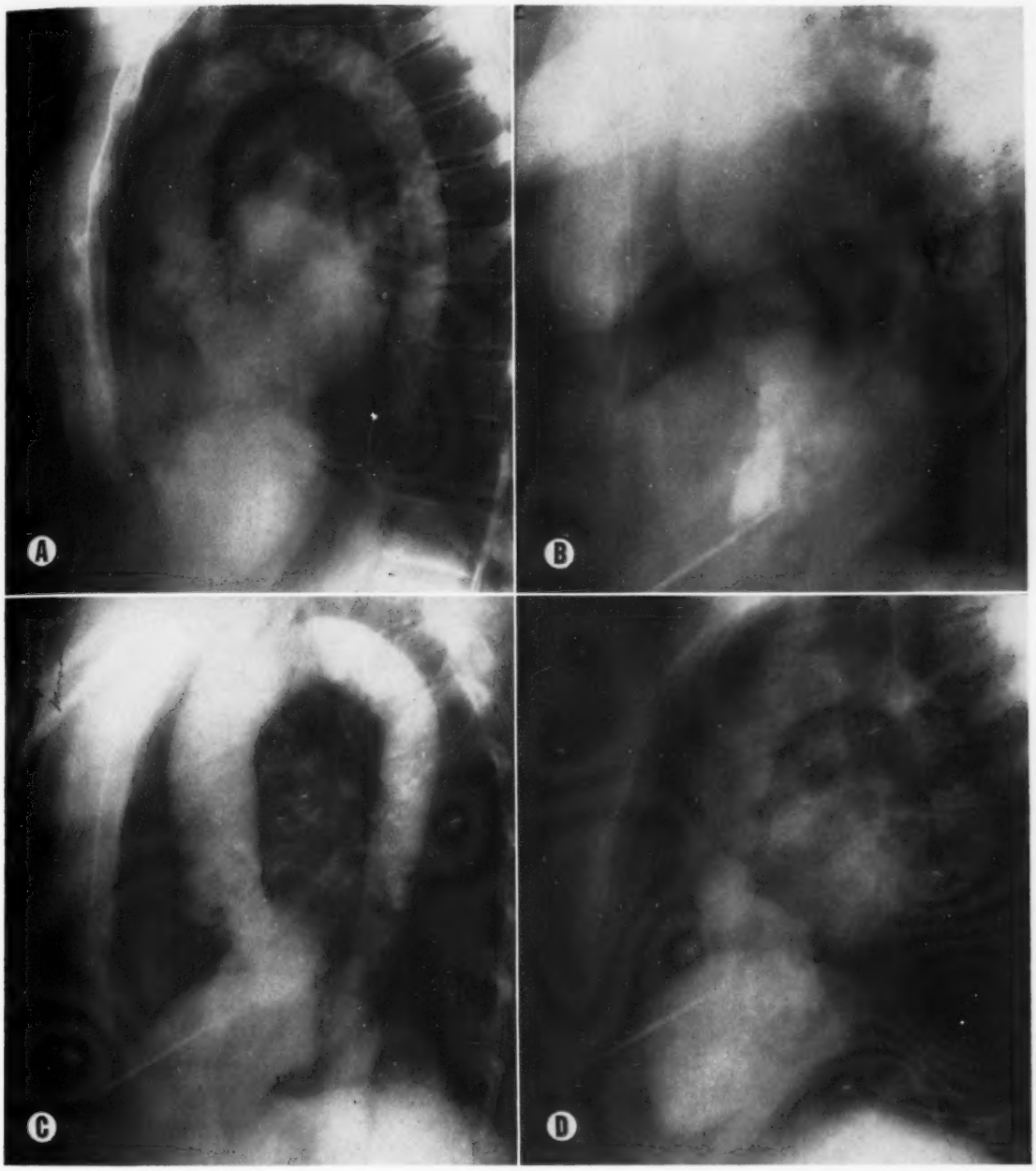

FIG. 10. Cardiac ventriculographies—left. Aortic valve visualization. (*A*) Case V. V. Normal aortic valve. Patient has mitral insufficiency. No evidence of aortic valvular disease at surgery (cross plication posterior commissure, mitral valve.) (*B*) Case F. A. Thickened aortic valve cusps. Verified by aortic valve commissurotomy for aortic stenosis. (*C*) Case T. M. Thickened, deformed aortic valve cusps. Deformed, stenotic aortic valve at aortic commissurotomy. (*D*) Case G. G. Thickened aortic valve cusps and grade I regurgitant opacification of left atrium. Proved as aortic stenosis, mitral stenosis and Grade 1 mitral insufficiency at surgery (aortic and mitral commissurotomies).

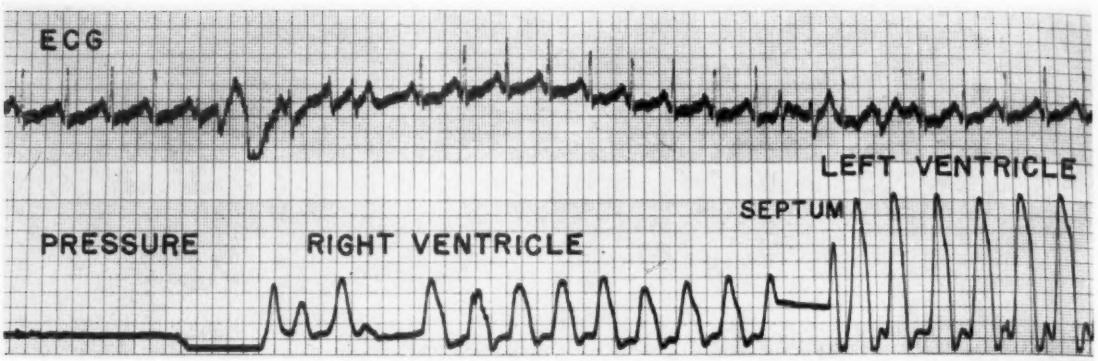

FIG. 11. Case J. N. Pressure and electrocardiographic tracings. Needle passage into right ventricle, through septum, into left ventricle. Right ventricle pressure 40/0–5; left ventricle pressure 115/0. Electrocardiogram shows ventricular premature beats as needle tip enters right ventricle and as septum is traversed.

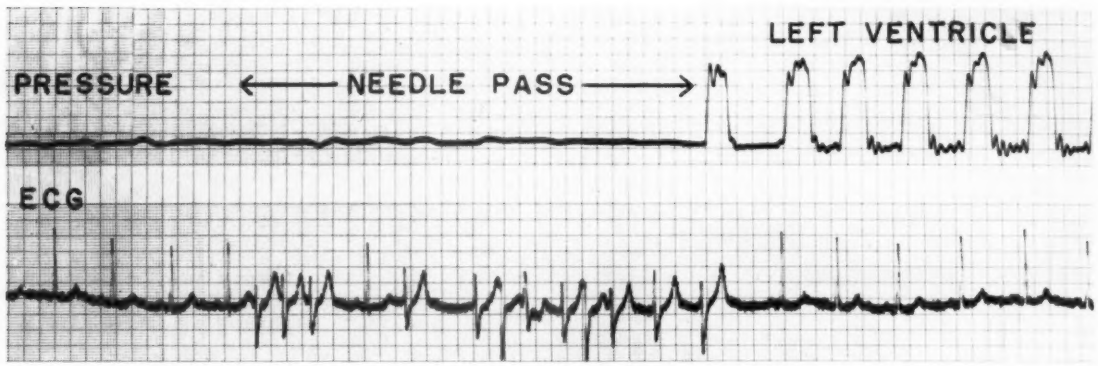

FIG. 12. Case M. H. Pressure and electrocardiographic tracings. Needle passage direct to left ventricle. Left ventricular pressure 140/0. Electrocardiogram shows prolonged ventricular tachycardia on slow passage of needle to the left ventricular chamber.

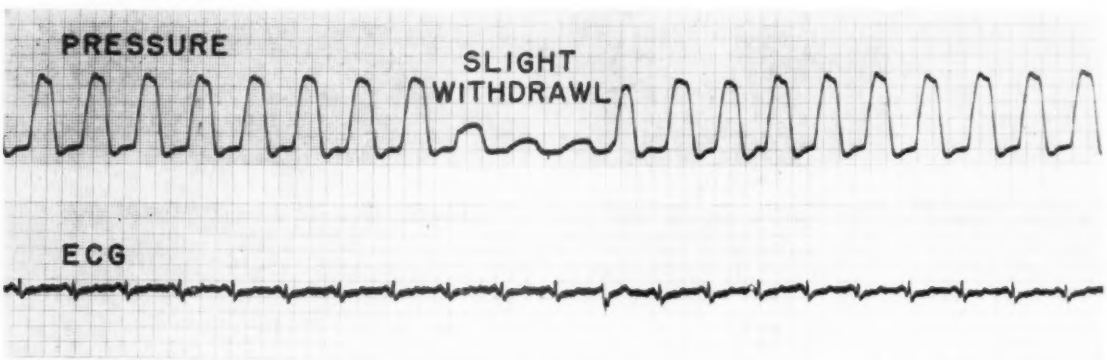

FIG. 13. Case L. B. Pressure and electrocardiographic tracings. Needle adjustment in ventricular chamber. Damping of left ventricular pressure waves as needle is slightly withdrawn. Return to satisfactory wave form with slight re-advancement of needle.

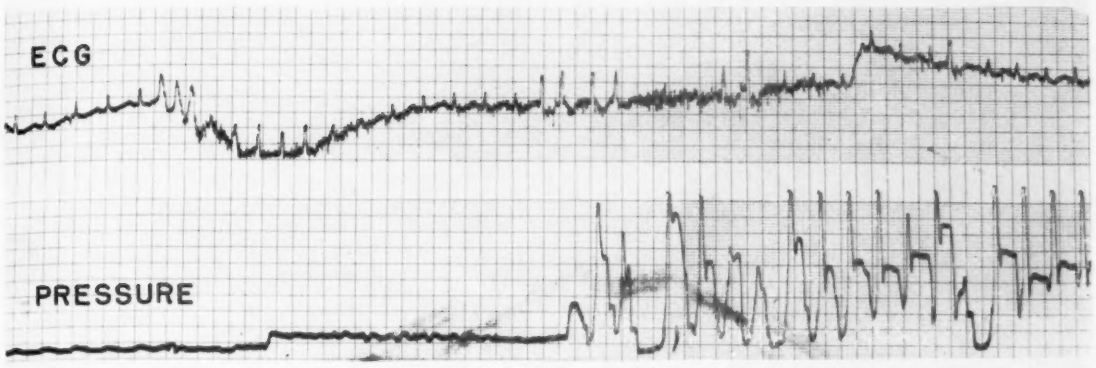

FIG. 14 Case J A. Pressure and electrocardiographic tracings. Damped left ventricular pressure waves. During the latter portion of ventricular systole and the succeeding diastole, the curve is damped, indicating that during this phase of ventricular movement the needle tip is partially in the ventricular wall.

Three of these 8 cases showed striking electrocardiographic aberrations, although only one of the 3 was of more than temporary duration.

It is obviously essential that the preexamination electrocardiographic data be known, both from the standpoint of excluding those patients who have a serious arrhythmia of a nature contraindicating cardiac ventriculography and in evaluating the significance of electrocardiographic changes produced by the procedure.

The arrhythmias occurring during cardiac ventriculography, incident to both needle puncture and diodrast injection, as well as those occurring in the immediate postinjection period are recorded in Table II.

Ventricular Premature Beats. Ventricular premature beats represented the most frequently encountered arrhythmia during ventricular puncture and were observed in 46 of the 77 studies. These were frequent in occurrence in 17, and only occasional in the remaining 19 examinations. Needle passage across the ventricular septum resulted in ventricular premature beats in 13 cases, but never exceeded 3 beats. Ventricular prematures were observed in 29 examinations at the time of actual diodrast injection, and never exceeded 5 beats. These were thought to be related to endocardial stimulation by the jet of opaque medium against the opposing ventricular wall. Premature ventricular beats were observed in only 2 patients in the postinjection period.

Ventricular Tachycardia. All instances of 3 or more consecutive beats of ventricular origin were considered as ventricular tachycardia. This major arrhythmia occurred 44 times during ventricular puncture. More than one episode of ventricular tachycardia was recorded in 17 of these 44 studies. The septum was traversed in 34

TABLE II

DISTURBANCES OF CARDIAC RHYTHM INCIDENT TO CARDIAC VENTRICULOGRAPHY

	Needle Puncture	Diodrast Injection	Immediate Postinjection Period
Ventricular premature beats	46	29	2
Ventricular tachycardia	44	23	2
Ventricular flutter	1	1	0
Coupling	10	6	2
Bigeminal rhythm	3	1	0
Bradycardia	3	26	1
Tachycardia of supraventricular origin	0	1	23
Sinus arrest	0	2	0
Ventricular asystole	0	2	0
Conduction disturbances	5	7	1

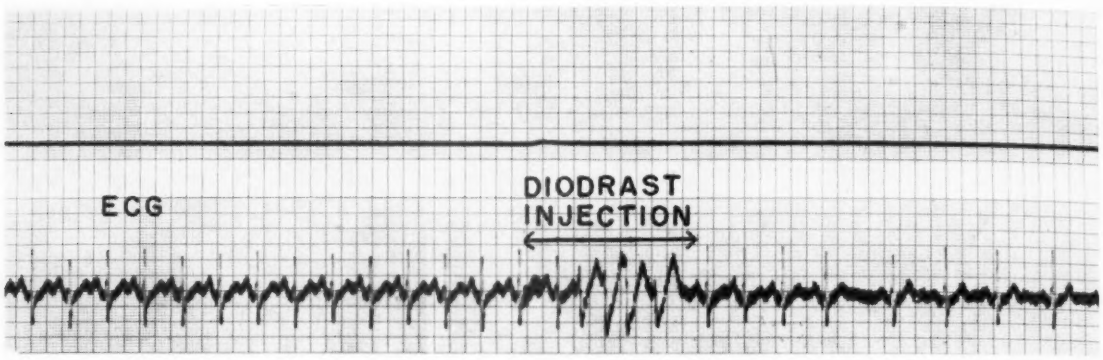

FIG. 15. Case M. S. Electrocardiographic tracing. Electrocardiographic changes immediately following diodrast injection. Occurrence of 4-beat ventricular tachycardia concurrent with diodrast injection. Prompt return to normal sinus rhythm, with a short period of bradycardia.

of the examinations in the entire series; ventricular tachycardia ensued at the septum in 8 of these 34 studies.

The duration of the tachycardia ranged between 3 and 5 beats in length, in 22 of the 44 examinations. A 6- to 9-beat ventricular tachycardia occurred in 16 examinations and a tachycardia of longer duration was observed in 6 of the studies. During diodrast injection, ventricular tachycardia occurred 23 times. Figure 15 shows the occurrence of a 4-beat ventricular tachycardia concurrent with diodrast injection. Multiple runs of tachycardia were observed in 4 studies in this group. A 3- to 5-beat ventricular tachycardia occurred in 14 of these 23 examinations, and a 6- to 9-beat tachycardia in the remaining 9 instances. It appeared in 2 studies during the postinjection period.

Ventricular Flutter. Ventricular flutter occurred in 2 examinations. Both of these were short-lived arrhythmias. One was incident to advancement of the needle through the myocardium; the second occurred at the time of diodrast injection.

Coupling. Coupled beats were noted in 10 examinations during ventricular puncture, in 6 during diodrast injection and in 2 during the postinjection period.

Bigeminal Rhythm. This was present for short periods during ventricular puncture in 3 studies, and occurred once at diodrast injection.

Bradycardia. Bradycardia ensued as the needle passed through the myocardium in 3 examinations. It occurred in 26 of the studies, incident to diodrast injection. The slowing occurred within 1 to 2 beats after the injection was begun. The periods of bradycardia varied from two to twelve seconds. The mechanism of this bradycardia is unknown, but because of its prompt appearance, a reflex origin is postulated. Due to its early timing, the afferent stimulus is thought to originate in the myocardium itself. The efferent stimuli suggest a strong vagal effect. Fifteen of the cases showing a bradycardia concurrent with diodrast injection had a normal sinus rhythm, and the slowing of the heart rate manifested itself as a sinus bradycardia. Twelve of the patients manifesting a bradycardia had a pre-examination rhythm of atrial fibrillation.

Tachycardia. One patient in the series developed a supraventricular tachycardia during diodrast injection. During the postinjection period, tachycardia occurred in 22 instances. Seventeen of these followed after a period of bradycardia. In the remaining 5, there was no significant slowing of the heart rate immediately preceding the development of the tachycardia. The increase in heart rate varied from 16 to 83 per minute, with an average increase of 29 beats per minute.

Sinus Arrest. Actual transient sinus arrest was precipitated on 2 occasions, both at diodrast injection.

Ventricular Asystole. Transient ventricular asystole occurred in 2 patients, both of whom showed a pre-examination rhythm of atrial fibrillation.

Conduction Disturbances. The conduction disturbances produced by cardiac ventriculography are presented in Table III. These occurred with ventricular puncture on 5 occasions. All were temporary, and spontaneous resumption of normal conduction followed. Similar arrhythmias were observed in 7 patients simultaneously with the diodrast injection. These were reversible except for one case (no. 43—Table 1). In this case there was inadvertent injection of some of the diodrast into the upper portion of the interventricular septum, and when the diodrast was injected ventricular asystole occurred, persisting for 6.5 seconds. During this interval, the *P* waves were present at the rate of 130 per minute. Complete atrioventricular dissociation followed, with a ventricular rate of 37 per minute. Shortly thereafter, a 3 to 2 block developed, followed by Wenckebach's phenomenon. Complete heart block recurred and has persisted for four months to the time of the present report. One patient developed atrioventricular dissociation in the postinjection period, but this was a temporary arrhythmia.

ST segment and *T* wave changes occurred in 3 cases. The *ST* segment elevation was observed at ventricular puncture in one patient, and this change persisted through the time of diodrast injection and for a short period thereafter. In the postinjection period, *ST* segment elevation occurred in one examination. *T* wave changes were absent in this case. In a third patient, *ST* segment depression and *T* wave inversion were observed in the postinjection period. These changes were transitory and, in the latter case, may represent relative myocardial anoxemia.

Table III

CONDUCTION DISTURBANCES INCIDENT TO CARDIAC VENTRICULOGRAPHY

Incident to Ventricular Puncture
Fusion beats; A-V conduction disturbance
A-V dissociation
Right bundle branch block; delayed intraventricular conduction
Nodal escape, 1-beat; wandering pacemaker
Nodal escape, 1-beat
Incident to Diodrast Injection
Changing aberrant conduction through bundle
Right bundle branch block
Second degree heart block
Nodal rhythm; first degree heart block
Ventricular escape—1 beat; nodal rhythm
3 to 2 block; Wenckebach's phenomenon; complete heart block
Nodal escape, 1-beat
During Postinjection Period
A-V dissociation

QUALITY OF EXAMINATION

The examination was considered to be a "failure" if satisfactory opacification of the designated ventricle was not obtained. On this basis, of the total of 77 examinations we classified 13 as failures (see Table 1). Causative factors considered incident to these failures were: Unintentional injection of the wrong ventricle in 4 examinations, in 2 of which there was partial intramyocardial injection; needle tip too high in the outflow tract of the ventricle for satisfactory opacification in 3; intramyocardial and/or intrapericardial injection in 3; syringe breakage with resultant delivery of an inadequate amount of diodrast to opacify the ventricle in one; failure of seriographic equipment during one examination, and improper positioning for satisfactory filming of one patient.

Excluding the 13 failures, the quality of the contrast opacification of the ventricle was adjudged to be excellent in 32 examinations; good in 21; fair in 7, and poor in 4 of the series.

The classification "poor" was applied to those studies in which, while the ventricle was opacified, the degree of contrast with the remainder of the heart was low. Classification of "poor" was for the following reasons: Needle tip too high in outflow tract

of ventricle; inadvertent partial intramyocardial injection; syringe breakage resulting in delivery of only 10 cc. of diodrast, and the fourth patient was of unusually large size habitus.

All of the examinations, including those classified as fair and poor but excluding the failures, provided information which we considered to be sufficient for our purposes, even though the films of the fair and poor groups were not of good contrast quality.

REACTIONS AND COMPLICATIONS

The reactions to cardiac ventriculography, in our experience, are quite similar to those occurring with angiocardiography, except that for *left* ventriculography they are usually somewhat more pronounced. The subjective sense of heat and the flushing are more profound but have not, in our experience, been of alarming degree. The rather frequent occurrence of a short period of bradycardia concurrent with diodrast injection, and the following period of tachycardia have been previously noted but produce no subjective symptoms. Occasionally, nausea and, infrequently, vomiting have been observed, as with angiocardiography. We have noted the occurrence of slight to moderately severe headache in somewhat less than half of our series of patients.

Right ventriculography has produced about the same intensity of reaction as angiocardiography.

Excluding the patients who have had an inadvertent intramyocardial injection, there has been no complaint of substernal or chest pain, except for a few patients who gave evidence of distress of a nature which we considered to be due to hemopericardium.

There have been no known instances of pneumothorax in our series.

Excluding the cases with inadvertent intramyocardial injection of diodrast, we have had but one patient who exhibited serious shock. This patient had a successful left ventriculography and experienced no discomfort until approximately ten minutes following injection, at which time he rather precipitately went into a state of severe shock, requiring oxygen administration and 5 per cent glucose venaclysis with levophed. This shock-like state persisting for twelve hours was followed by complete recovery. It is our belief that this patient exhibited a true anaphylactic reaction to the diodrast. His routine intradermal diodrast test, however, was negative.

Cardiac surgery was performed on 35 patients following cardiac ventriculography. The time lapse between ventriculographic study and surgery was one day for 4 patients, two to four days for 9 patients, five to eight days for 17 patients, and nine to thirteen days for 4 patients. There was a twenty-day interval between ventriculography and surgery in one patient.

At the time of cardiac surgery, note was made of the occurrence of any bloody intrapericardial fluid. Eleven patients demonstrated an estimated 50 to 100 cc. of bloody fluid, two had from 100 to 200 cc., and one had an estimated 300 cc. One patient exhibited about 400 cc. of bloody fluid in the pericardial sac, but had had a left heart catheterization study involving left atrial needle puncture four days subsequent to left cardiac ventriculography, and six days prior to surgery. The remainder of the operated patients showed no evidence of bloody fluid within the pericardium. Two patients exhibited some ecchymosis of the pericardial fat pad.

In the entire series of 77 cardiac ventriculographies, no clinical evidence of pericardial tamponade was encountered. Of those patients who had cardiac surgery through the left hemithorax and in whom the pericardium was opened to permit of direct inspection of the surface of the heart at the site of myocardial needle puncture, one patient presented an area, approximately 4.0 cm. in diameter, of ecchymosis and somewhat brownish discoloration of the myocardium. The other patients having such exploration presented no visible external evidence of myocardial trauma.

There has been no known instance of needle laceration of a coronary artery or coronary branch vessel in this series.

Intramyocardial injection of diodrast accidentally occurred in 8 examinations. In one of these there was combined intramyocardial and intrapericardial infiltration of the entire dosage of 30 cc. of diodrast. This patient's electrocardiogram (lead II) showed frequent ventricular premature beats at diodrast injection, with a return to normal rhythm in the immediate postinjection period. The patient experienced slight pain but displayed no shock symptoms.

All of the other 7 cases showed at least some portion of the injected opaque substance within the ventricular cavity. In 2 of these the amount of diodrast in the myocardium was very minimal. Neither of these patients experienced either pain or shock. The electrocardiographic observations were almost identical in these 2 cases, there being a 6-beat and 7-beat ventricular tachycardia, respectively, and occasional coupling at diodrast injection with prompt return to normal rhythm in the immediate postinjection period.

Four patients had a moderate amount of diodrast injected into the myocardium. The first of these 4 experienced mild pain and mild shock. At injection, there was a 9-beat ventricular tachycardia and a short run of ventricular flutter, with postinjection electrocardiographic record showing frequent coupling. The second patient had substernal pain and shock which persisted for twenty-four hours, after which there was recovery. On needle puncture, atrioventricular dissociation was recorded and this continued through the injection period. Postinjection electrocardiogram showed *ST* segment elevation. The third patient had slight pain but no shock. The electrocardiogram recorded a right bundle branch block and delayed intraventricular conduction on needle puncture, and at injection a changing aberrant conduction through the bundle. Immediately postinjection, an atrioventricular dissociation was recorded. This changed to an upper nodal rhythm in fifteen minutes. Normal sinus rhythm was recorded twenty-four hours later. The immediate postinjection electrocardiogram of this patient is shown in Figure 16. Surgery directed toward correction of mitral and aortic stenosis (mitral and aortic commissurotomies) two days later revealed no gross evidence of myocardial damage. The diodrast infiltrating the posterior wall of the left ventricle is seen in Figure 17.

The fourth patient, who exhibited some palpitation but no pain or shock, presented electrocardiographic evidence of coupling, changing to a bigeminal rhythm, at needle puncture. At diodrast injection, this changed to a supraventricular tachycardia with aberrant conduction and a right bundle branch block. These electrocardiographic changes persisted through the immediate postinjection period.

We have encountered one serious complication among the 8 cases of accidental intramyocardial injection. This patient, who had had clinical evidence of mitral valvular insufficiency, experienced neither pain nor shock as a result of the procedure. The electrocardiographic recording, however, revealed the development of complete heart block simultaneous with the diodrast injection. Figure 18 reveals a fair-sized globule of diodrast high in the interventricular septum. This complete heart block has persisted and is now of four months' duration. During this period, the heart size has increased, and it is our belief that the existing mitral insufficiency has been aggravated by the mechanical inefficiency resulting from the complete heart block.

All of the occasions of intramyocardial and/or intrapericardial infiltration of diodrast occurred among the first 47 cardiac ventriculographies, and we have experienced no frank infiltration complications among the last 30 examinations. We believe that we have been able recently to avert the complication of infiltration by meticulous attention to the following points of technique:

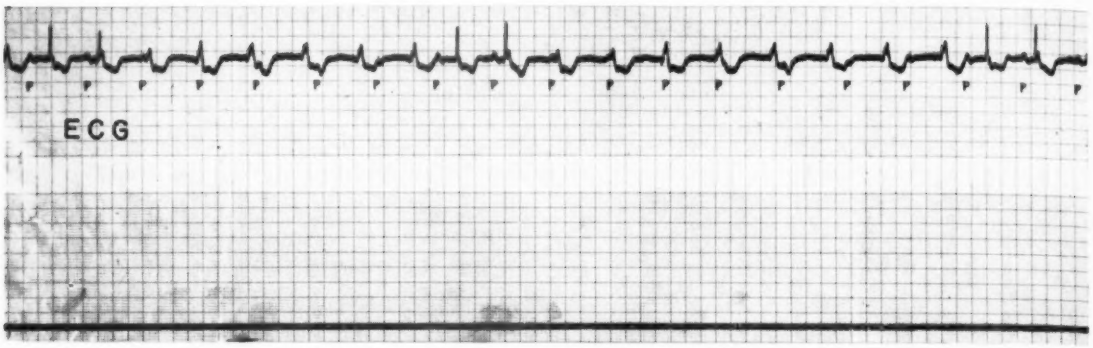

FIG. 16. Case H. B. Electrocardiographic tracing. Electrocardiographic changes immediately following inadvertent intramyocardial diodrast injection (partial). Patient experienced slight pain but no shock. Atrioventricular dissociation immediately following diodrast injection. (This changed to upper nodal rhythm in fifteen minutes. Normal sinus rhythm re-established within twenty-four hours.) See Figure 17 for film representation of intramyocardial injection. Surgery for mitral and aortic stenosis (mitral and aortic commissuortomies) two days later revealed no gross evidence of myocardial damage.

1. *Aiming of Needle.* Our objective is to locate the needle tip (for left ventricular puncture) approximately half-way between the mitral valve and the apex of the heart. For the right ventricle, our objective is the center of the right ventricular cavity. Determination of these points and the direction of needle insertion are materially aided by careful inspection of the initial scout film with its series of lead numbers in the line of the long axis of the ventricle (see Section on Roentgenographic Tech-

FIG. 17. Case H.B. Inadvertent intramyocardial injection (partial). Patient experienced slight pain but no shock. Diodrast infiltration posterior wall of left ventricle. (See Figure 16 for electrocardiographic changes.)

FIG. 18. Case R. H. Inadvertent diodrast infiltration into interventricular septum. Fair-sized globule of diodrast seen high in interventricular septum. Patient developed complete heart block, persisting to present (four months).

nique). It is our belief, particularly in view of the experience with the case of infiltration of the septum and the development of heart block, that it is wiser to err on the side of low position of the needle point rather than of a position related to the upper portion of the septum.

2. *Adjustment of the Needle Point.* The maneuver of gradual withdrawal of the needle, after first obtaining a ventricular pressure tracing, until the pressure tracing becomes damped (see Section on Technique of Ventricular Puncture and Injection) and then re-advancement of the needle 0.5 cm., or until a good ventricular pressure wave is recorded, is important in avoiding septal or ventricular wall infiltration.

3. *Patient Breathing.* Fully-comprehended instructions are given to the patient to the effect that he must avoid taking a deep breath during the entire puncture and injection procedure. We believe that lack of careful and comprehensive instruction and /or nonobservance of the detail of shallow breathing were responsible for at least several of our infiltration complications.

4. *Non-Motion of Needle or Patient.* Further positioning or moving of the patient after intracardiac insertion of the needle must be strictly avoided. By the same token, no inadvertent movement of the needle should be permitted.

If there is any question as to the strict adherence to the foregoing points of technique, or if a completely "undamped" pressure wave pattern is not obtained, the needle should be withdrawn or re-manipulated.

CONTRAINDICATIONS TO CARDIAC VENTRICULOGRAPHY

Our experience has not, in our opinion, been sufficiently extensive to justify positive conclusions regarding contraindications to the procedure. We do not, at this time, believe that chronic atrial fibrillation or flutter contraindicates cardiac ventriculography. However, atrial fibrillation with a poorly controlled ventricular rate or paroxysmal atrial tachycardia would seem, in our opinion, to be contraindicative.

Atrial tachycardia due to vagal release from atropine administration is, we believe, another contraindication.

Ventricular tachycardia, either spontaneous or induced by medication, is also considered by us as a contraindication to cardiac ventriculography.

We do not consider pre-examination occurrence of ventricular premature beats as contraindicative.

We have not employed cardiac ventriculography in children or infants, and would be extremely reluctant to do so, as we believe the possibility of intramyocardial injection of the opaque medium is of considerably greater likelihood with a small-sized ventricular cavity.

SUMMARY

1. The term "cardiac ventriculography" is employed in this presentation to designate a procedure of direct transthoracic needle puncture of the heart for the purpose of effecting opacification of the left or right ventricle.

2. The application of direct ventricular opacification to the study of certain pathologic states of the ventricles, the atrioventricular valves and the thoracic aorta is discussed.

3. An analysis of an experience with 77 cardiac ventriculographies performed in 60 patients is presented.

4. The details of roentgenologic technique, ventricular puncture, pressure and electrocardiographic monitoring, and opaque medium injection are discussed.

5. The intracardiac pressures observed and the electrocardiographic changes incident to the procedure are presented.

6. The reactions to the procedure, the occurrence of inadvertent intramyocardial and intrapericardial infiltration of the opaque substance in 8 patients, are reported as is our approach to the avoidance of these complications.

7. An evaluation of the quality of the cardiac ventriculographies based upon the

degree of contrast visualization of the ventricle is tabulated.

8. Possible contraindications to cardiac ventriculography on the basis of our experience with this series are suggested.

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THE VALUE OF FAST ANGIOCARDIOGRAPHY IN THE EARLY DIAGNOSIS OF PATENT DUCTUS ARTERIOSUS*

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THE problem of diagnosis of patent ductus arteriosus is intimately connected with the surgical treatment since most cardiologists and heart surgeons agree that an uncomplicated patent ductus arteriosus is an indication for surgery.[7,25,34,35]

The surgical problem of patent ductus arteriosus is a question of early diagnosis. In 1944, Crafoord, *et al.*,[7] stated that patent ductus arteriosus should be surgically corrected in early childhood but that one should wait if possible until the child had passed its third birthday. In recent years some investigators have recommended surgical correction in still younger subjects in severe cases, because in so many instances of isolated patent ductus arteriosus, death may occur in infancy due to the malformation[23] and because of the low incidence of spontaneous closure of a pathologically patent ductus.[44]

The incidence of death in patent ductus arteriosus in infancy has been recorded to be as high as 25 per cent in some series. This is, however, a difficult fact to establish since pathologists usually consider the condition a normal finding in infancy regardless of its size in relation to the aorta.[2] Many cases have been successfully operated upon before one year of age and in some of these surgery has been considered life-saving.[2,23,32,44]

In infants and young children the clinical diagnosis, however, is often difficult to establish.[1,17,42] The usual clinical findings —thrill, accentuated pulmonary second sound, high pulse pressure, peripheral vascular manifestations, palor, stunting of growth, etc.—are missing in a rather large percentage of cases. Even the most important clinical sign, the continuous murmur may be absent and if a murmur is present, it may be short and systolic,[1] probably due to the special pressure distribution in the aorta and pulmonary artery during the first years of life. Not until after the diastolic pressure in the systemic circulation has become higher than in the pulmonary circulation does the machinery murmur develop.

The diagnosis of patency of the ductus arteriosus in the absence of a continuous murmur is still more difficult. Twenty-four patients suspected of having a patent ductus arteriosus without the continuous murmur have recently been reported by Dammann and Sell.[8] Nineteen of the patients were under five years of age. The examination included roentgenoscopy, roentgenography, electrocardiography, and in 17 cases cardiac catheterization. Only 15 of the 24 patients were proved to have a patent ductus arteriosus. Two patients had aortic septal defects and 7 were diagnosed at surgery as having either high ventricular septal defects or Eisenmenger's complex. The cardiac catheterization proved of real aid in the diagnosis but was not infallible. The authors conclude by stressing the need for more accurate diagnosis. The 13 patients with patent ductus arteriosus who survived surgery were greatly benefited. Those in heart failure improved rapidly and all are leading normal active lives.

The importance of establishing the diagnosis at an early age is stressed by several authors. In Ekström's[13] clinical study of 290 cases of open ductus arteriosus, the diagnosis of congenital heart disease was made in 28 per cent during the first year.

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The methods of examination in these 290 cases included auscultation, or recording of the machinery murmur, in 95 per cent and in the remaining 5 per cent heart catheterization, thoracic aortography and exploratory thoracotomy. Intravenous angiocardiography is not mentioned. This is rather surprising since angiocardiography may be of value in the preoperative assessment of a patent ductus arteriosus by helping to rule out associated congenital defects.

Retrograde aortography opened a new field in the diagnosis of patent ductus arteriosus, permitting direct demonstration of the open ductus (Castellanos, 1939; Jönsson and co-workers, 1948[21]). In adults and young children the technique is unfortunately quite difficult, but it is possible—as Keith[23] has shown—to carry it out in infants with excellent results. An artery must, however, be sacrificed.

A limitation of the method is that it cannot give full information about the presence of other associated congenital malformations of the heart, so necessary in assessing the operability of the actual case. The following table of Boesen and Wendel,[4] illustrates the poor prognosis of some common congenital malformations (Table 1). It answers the question of whether the coexistence of septal defects worsens the prognosis in cases of patent ductus arteriosus and it also brings out the necessity of early operation.

Intravenous angiocardiography, when carried out with precision, is a very valuable method, which can be used successfully in those infants who have manifest heart disease (enlarged heart, dyspnea, peripheral edema) and in whom the diagnosis is not obvious after employing the routine methods of examination.

INTRAVENOUS ANGIOCARDIOGRAPHY

In the presence of a patent ductus, blood is shunted from the aorta into the pulmonary artery and it was therefore anticipated that by intravenous angiocardiography the ductus itself might be visualized by contrast medium passing into the pulmonary artery from the aorta. This expectation, however, has not been realized,[12] except in occasional cases. The ductus has a hidden position and is therefore difficult to observe.

The routine use of two simultaneous projections increases the possibility of demonstrating the patent ductus arteriosus which according to our experience, is best visualized in the left anterior oblique position.

TABLE I

AGE DISTRIBUTION FROM AUTOPSY MATERIAL OF SOME TYPES OF CONGENITAL HEART DISEASES IN INFANCY
(BOESEN AND VENDEL, 1953)

	Total Number	Number of Infants Living at an Age of				
		9 days	1 month	7 months	1 year	4 years
Patent Foramen Ovale (P. F. O.) (with symptoms of congenital heart disease)	6	2	0			
Atrial Septal Defect (A. S. D.)	15	13	12	5	3	0
Ventricular Septal Defect (V. S. D.)	46	38	32	6	3	0
Patent Ductus Arteriosus (P. D. A.)	17	13	8	3	3	0
P.F.O.+V.S.D.	17	16	16	2	2	0
P.F.O.+P.D.A.	15	3	3	1	0	
A.S.D.+V.S.D.	2	2	2	0		
A.S.D.+P.D.A.	12	7	30	0		
V.S.D.+P.D.A.	12	6	33	0		
P.F.O.+V.S.D.+P.D.A.	13	11	9	1	0	
A.S.D.+V.S.D.+P.D.A.	3	3	2	0		

The decisive limitation of the method using intravenous injection is the *poor concentration of the contrast medium in the left heart and aorta*, so that the open ductus in many cases cannot be demonstrated either directly or indirectly with precision. This is true in older children and adults, where usually the diagnosis is evident clinically.[42] In infants and younger children the opacification of all the heart chambers and aorta is generally sufficient to establish the diagnosis even if the ductus itself cannot be seen. In a large group of these children with presumptive or doubtful clinical signs of patent ductus arteriosus, the diagnostic problem has, according to our experience, been solved with the aid of intravenous angiocardiography.

Moreover, by using this method, especially the direct fast angiocardiography with synchronous electrocardiographic recording, conclusions concerning pathophysiologic function of the heart may, to a certain extent, be permitted.

Roentgencinematography offers an advanced approach to the study of the cardiac dynamics. The high speed of pictures permits a continuous recording of the hemodynamic events during one and the same cardiac cycle. It is even possible to accurately time the occurrence of the diastolic shunt (Fig. 7). Bidirectional shunts can also be well demonstrated in cases complicated by pulmonary hypertension (Fig. 6).

The radiation exposure of the patient is cut down to a permissible level by using image intensification. The recording on film implies the advantage of the combination of both frame by frame in a moviescope and by repeated projections as an endless loop.

DIRECT DEMONSTRATION OF THE OPEN DUCTUS ARTERIOSUS

In cases of open ductus arteriosus, combined with other congenital malformations of the heart where the contrast medium does not become diluted in the lungs before it reached the aorta, there is greater possibility of demonstrating the open ductus directly. Examples of this are tetralogy of Fallot, transposition of the great vessels, etc. (Fig. 1).

It is well known that the pulmonary arterial pressure may increase to such an extent that it exceeds the aortic pressure, and, consequently, blood flows through a patent ductus from the pulmonary artery to the aorta. Several such cases are published.[29] This may happen in newborn infants as the ductus arteriosus remains patent during the first three to four days of life and there is a moderate pulmonary hypertension. The right ventricle is as well developed as the left. Normally there is a significant left to right shunt, but a reversal sometimes takes place. We have demonstrated this fetal direction of the shunt in 4 newborn infants with asphyxia. A typical angiocardiogram of such a case is shown in Figure 2. In this instance the explanation of the reversal of the blood flow may have been atelectasis of the lungs.

INDIRECT ROENTGENOLOGIC SIGNS OF PATENT DUCTUS ARTERIOSUS

There are certain roentgenologic signs, called indirect or secondary signs, which suggest more or less strongly the presence of a patent ductus arteriosus. These signs are the result of both anatomic alterations and pathophysiologic disturbances. Each sign has a relative value and they must be considered together in arriving at a diagnosis. All, except the first one, are dependent on the blood flow through the open ductus. Provided that the open ductus is sufficiently large, some of these indirect signs may be seen at roentgenoscopy and during routine roentgenographic examinations. They are demonstrated far more strikingly, however, by angiocardiography.

The most important indirect signs of patent ductus arteriosus are:

1. Aortic infundibular dilatation.
2. Dilatation of the pulmonary artery.
3. Elevation of the truncus—conus pulmonary artery and its left main branch.
4. Increased pulmonary artery output.

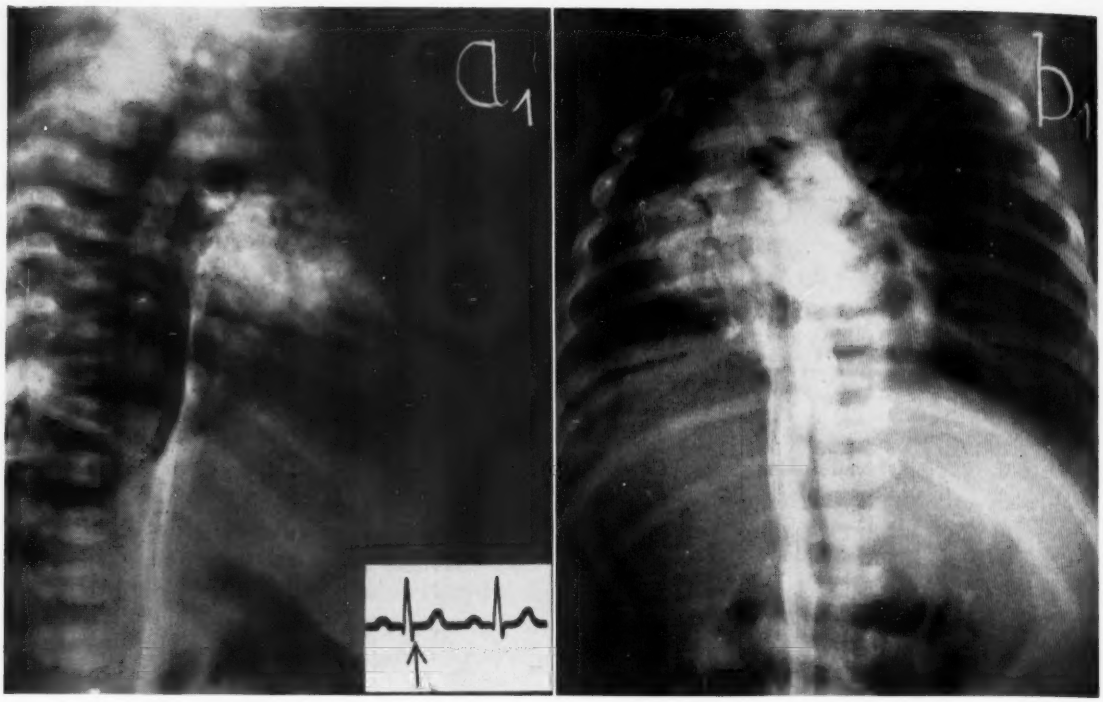

FIG. 1. *Direct demonstration of an open ductus arteriosus in a case of tricuspid atresia.* Right (a_1) and left (b_1) anterior oblique projections.

Ventricular systole. The right atrium and left ventricle are visualized, as well as the broadened aorta and pulmonary artery. The open ductus is clearly seen in both projections. Its existence and atypical course were verified at autopsy.

Increased filling of the central pulmonary blood vessels.

5. Dilution of the contrast medium in the pulmonary artery, best seen during ventricular diastole.
6. Prolonged circulation of the contrast medium in the pulmonary vessels.
7. Enlargement of the left atrium and ventricle.
8. Refilling of the pulmonary artery in the later phase of angiocardiography.

If a patent ductus arteriosus is complicated by *pulmonary hypertension,* indications of right ventricular enlargement and hypertrophy appear. An upturned apex is seen in the anteroposterior view, and in the lateral view an increased contiguity of the surface of the heart to the thoracic wall occurs. The vascularity of the lungs is not increased, and in the periphery of the lungs the vessels are narrow.

A localized dilatation of the descending aorta on its anterior aspect just beyond the isthmus was pointed out by Steinberg, Grishman and Sussman[10] and later by Furman.[15] The altered segment varies in size and shape from a localized bulge to a uniformly dilated segment. The appearance, however, is not pathognomonic. Dotter and Steinberg[12] found this localized dilatation repeatedly without evidence of a patent ductus arteriosus and they also noted its absence in proved cases of this disease.

In the left anterior oblique position the posterior border of the left main branch may create the illusion of a local dilatation if the penetration of the roentgen rays is too low. But the existence of a clear infundibular dilatation of the aorta in this region must be considered as one of the strongly suggestive indirect signs furnished by direct angiocardiography.

In conventional anteroposterior films Jönsson and Saltzman[22] observed a deformity of the outline of the aorta charac-

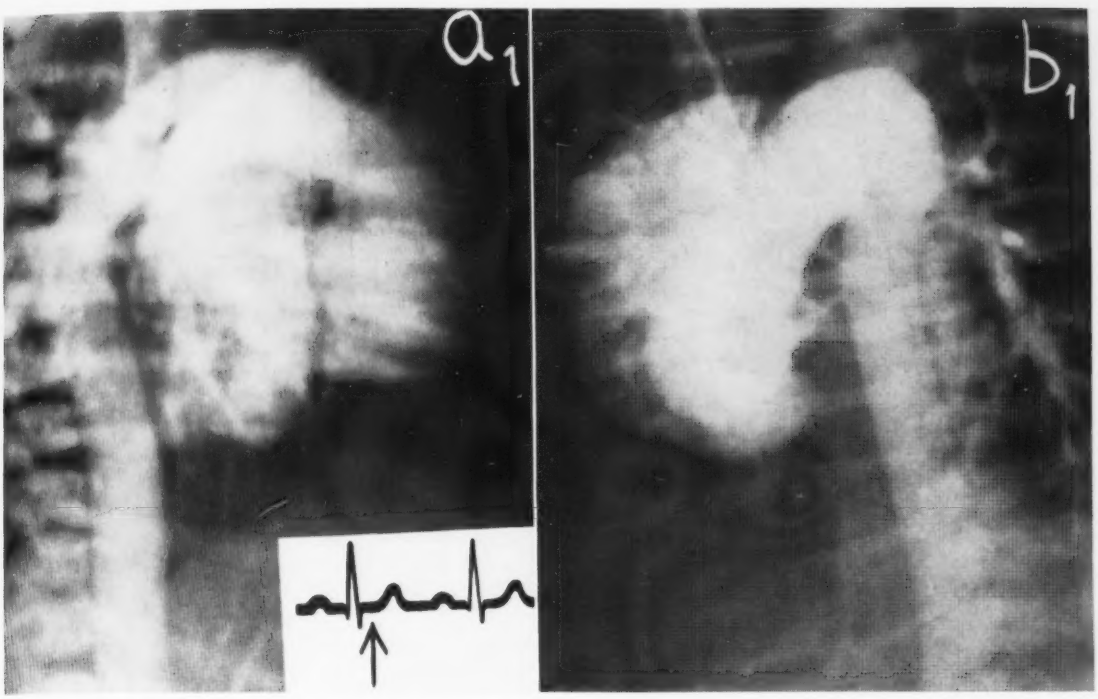

Fig. 2. *Patent ductus arteriosus with persistence of fetal type of circulation.* Synchronous angiocardiograms in right anterior oblique (a_1) and left anterior oblique (b_1) projection.

(a_1) The right atrium and right ventricle are well opacified as well as the pulmonary artery and the aorta descendens. The patent ductus is seen between the two main branches of the pulmonary artery.

(b_1) The right side of the heart is heavily filled with contrast material as well as the pulmonary artery and the aorta descendens. No medium is seen in the left side of the heart and the aorta ascendens is not visualized.

teristic of the infundibulum of the ductus in 21 of 39 patients with a patent ductus arteriosus. The deformity was absent in a series of normal subjects similarly examined. In a series of children studied together with F. Ulfsparre this sign was less frequently seen, and, unfortunately, this frequency decreased further the younger the child. It is difficult to obtain straight anteroposterior exposures when dealing with small restless children. In addition, many of the smallest children may have a large thymus which covers the upper part of the aorta, thus making it impossible to study the requisite portion of the aortic shadow.

One of the most outstanding features in patent ductus arteriosus is the *increased amount of blood that flows through the lungs*, sometimes without marked hypertension as would be expected, due to a remarkable power of *dilatation of the pulmonary artery and its branches*. This dilatation is clearly seen in direct serial angiocardiograms (Fig. 3). If the amount of blood flowing through the open ductus is constantly small, roentgenographic examination will show no such dilatation.

When the measurements are correlated with the heart phase during which the films were exposed, the maximum caliber of the pulmonary vessels is observed during ventricular systole. The minimum caliber is found near the end of the diastole, before the systolic output has begun.[28] At the beginning the dilatation is seen only in systole, but later it becomes permanent. The difference in caliber in diastole and systole ("hilar dance") is increased, but not markedly, and is generally not of the same degree as occurs in large atrial or ventricular septal defects with left-to-right shunts.

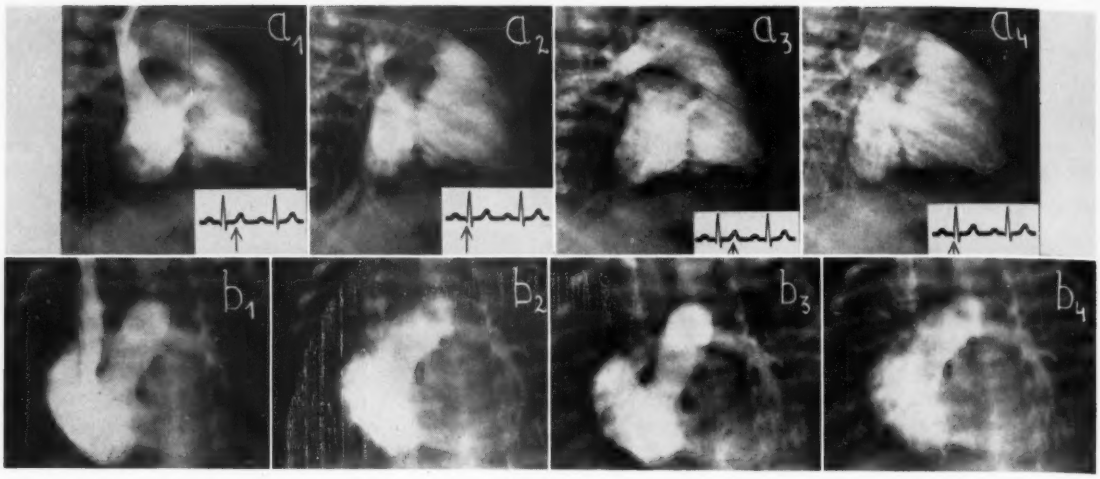

FIG. 3. *Diastolic dilution of the contrast medium in the pulmonary artery in patent ductus arteriosus.* Right (a_1–a_4) and left (b_1–b_4) oblique projections.

(a_1b_1) Starting ventricular systole. The pulmonary artery is well visualized.

(a_2b_2) End of ventricular diastole. The contrast medium is highly diluted in the stem of the pulmonary artery, but not in the branches of the vessel.

(a_3b_3) Following ventricular systole. The pulmonary artery is again intensely opacified.

(a_4b_4) In the next ventricular diastole dilution of the contrast medium in the stem of the pulmonary artery again takes place. The pulmonary artery is diminished in width.

This may be due to the "continuous" shunt from aorta to pulmonary artery, which is taking place during diastole as well as systole. In intracardiac left-to-right shunts the increase in the pulmonary flow is limited to the ventricular systole. The difference is demonstrated by the relative systodiastolic size of the pulmonary artery found in patent ductus arteriosus with a large shunt. An elongation of the pulmonary artery can also be seen as a result of the same process.

The elevation of the pulmonary artery and left main branch has been demonstrated by Steinberg, Grishman and Sussman.[40] The explanation of Mouqin, Durand and Hatt[31] that it is a result of aortic traction on the pulmonary artery may be correct.

A patent ductus, like an arteriovenous fistula, causes short circuiting of a certain amount of blood. The blood shunted from the aorta leads to a considerably *increased filling of the central pulmonary blood vessels* (Fig. 3).

The shunt from the aorta can be indirectly demonstrated by an evident *loss of contrast of the pulmonary artery*, a consequence of the dilution of the contrast medium by the blood which comes from the aorta. This indirect sign, described also by Goetz,[18] was manifest in nearly all of our cases and was quite obvious when using a rapid sequence of films which shows a sufficient number of different phases during the same heart cycle (Fig. 3 and 4). Observations of such phenomena as dilution or increased concentration of the opaque medium requires a continuous sequence of films, in which the entire hemodynamic mechanism is recorded in relation to the cardiac cycle.

During ventricular systole the contrast medium from the right ventricle usually produces a good and homogeneous opacification of the pulmonary artery. Sometimes a defect in the contrast-filled pulmonary artery can be seen at the entrance of the ductus—the larger the shunt, the larger the defect. A rough estimation of the size of the shunt in relation to the output of the right ventricle can thus be made. In ventricular diastole the dilution of the contrast medium in the pulmonary artery is greater than during ventricular systole,

when the pulmonary valves are closed allowing the medium to leave the right ventricle. It can also be noted that the dilution is greatest at the end of diastole. The disappearance of the medium from the pulmonary artery during ventricular diastole is typical of patent ductus arteriosus and aortic septal defect and is not seen in high ventricular septal defect. The localization of the site where the dilution is maximal may be of help in the differential diagnosis between the two conditions. When the shunt is small, a little filling defect is seen at the site of the entrance of the ductus arteriosus in the pulmonary artery.

The continuous murmur associated with patency of the ductus has been thought to be due to a continuous flow of blood through the ductus from the aorta into the pulmonary artery, depending upon a difference in the systolic and diastolic pressures between the systemic and pulmonary circulations. In newborn infants and in early infancy the systemic pressure is low and the pulmonary arterial pressure is relatively high (Hamilton, *et al.*, 1937). With the growth of the infant the systemic pressure rises and it has been suggested that the pulmonary arterial pressure falls gradually during the first six to seven months of life (Civin and Edwards, 1951). It has thus been thought that the blood flow at first will occur only during systole and the murmur will be purely systolic. When the diastolic pressure in the aorta becomes greater than the pulmonary diastolic pressure, the blood flow through the patent ductus will be continuous and the murmur will assume its characteristic quality. Normally this murmur appears at about the age of two to three years. This theory is not in accordance with the findings by angiocardiography insofar as a diastolic shunt can be present although it may not produce a murmur—a finding which we have stated several times.

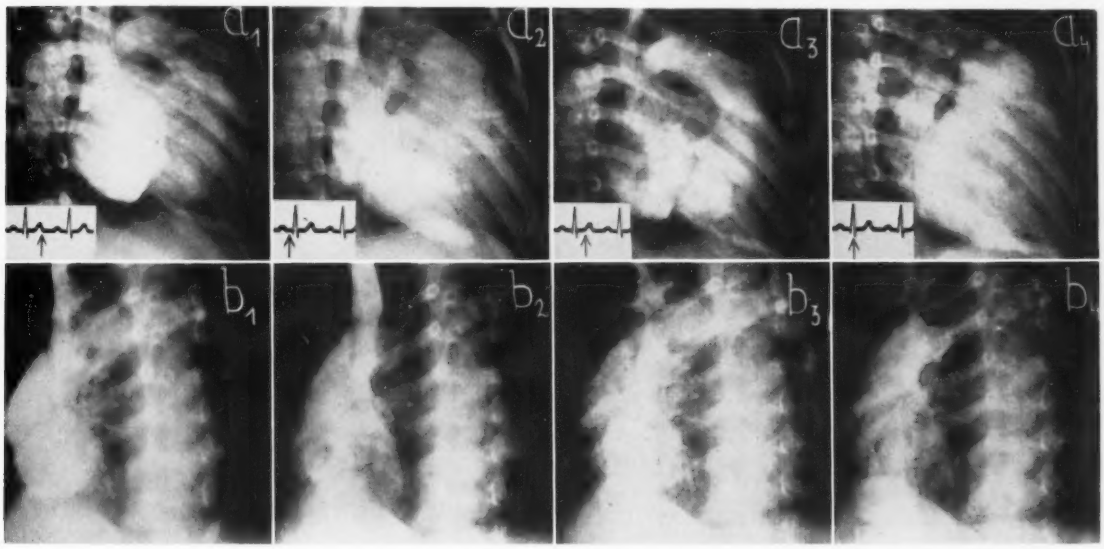

Fig. 4. *Dilution of the contrast medium in the pulmonary artery demonstrable during each ventricular diastole.* Right (a_1-a_4) and left (b_1-b_4) anterior oblique projections.

(a_1b_1) Ventricular systole. The pulmonary artery is well visualized in both projections.

(a_2b_2) End of the following atrial systole. The medium has disappeared from the pulmonary artery.

(a_3b_3) End of the ventricular systole. Together with a_1 it illustrates the characteristic way in which the right ventricle contracts. There is residual contrast medium at the base, the pulmonary conus is narrow, the pulmonary artery elongated and well opacified again.

(a_4b_4) End of the following atrial systole (isometric contraction phase of the ventricle). The contrast medium in the pulmonary artery is again "washed out" by the blood shunted into the pulmonary artery from the aorta during the ventricular diastole.

An important examination of diagnostic as well as prognostic value is the determination of the heart size. Systolic murmurs combined with increased heart volume have been the usual indication for angiocardiography in our cases. A large heart or an increasing relative heart size may be an important indication for operation of patent ductus arteriosus in infants. The size of the heart is most accurately estimated by a volumetric method which is simple and requires little time. Nomograms for calculation of the actual heart volume as well as for predicted heart volume and comparison with values from normal infants are available.[26] The widely used cardiothoracic ratio is unreliable. It involves a systematic error which may either accentuate or mitigate the pathologic tendency.[26]

More important than determination of the over-all heart size may be the determination of the size of a single chamber, the left atrium and ventricle especially, as neither the roentgenologic criteria nor the electrocardiographic findings are quite accurate for detection of left ventricular hypertrophy in this age group.[44]

The left heart must, when the peripheral circulation is constant, increase its stroke volume with the quantity of blood shunted into the pulmonary artery. By angiocardiography, *enlargement of the left atrium and ventricle* is directly shown. Due to the rounded form of the left atrium it is possible to estimate its volume by using two projections synchronously. Because of the great variations in size during the heart cycle, it is necessary to know in which heart phase the roentgenograms are taken in order to evaluate the atrial volume. The left atrium is enlarged only in cases in which there is fairly marked pulmonary vascular congestion. When hypertrophied, the left ventricle is covered by the right ventricle in a "peel-like" fashion seen in the left anterior oblique view, and best seen when the right ventricle is filled with contrast in diastole. The increased thickness of the wall of the left ventricle is directly seen in this projection. Here, too, the determination of the heart phase for each exposure from the synchronous electrocardiogram with automatic exposure marking facilitates the evaluation of variations in hypertrophy.

Prolonged or renewed visualization of the pulmonary artery has been expected in all cases but has not been found to be a reliable sign. By employing a fast injection technique, the right and left side of the heart are normally seen opacified successively, *i.e.*, the right side of the heart and the pulmonary artery are empty of contrast medium when the concentration of the medium is highest in the aorta. A drawback is that when the opacity has reached its maximum in the aorta there may still be some contrast medium left in the pulmonary vessels also in normal cases, and a prolonged or renewed visualization of the pulmonary artery is difficult to distinguish. In many cases of patent ductus arteriosus we have found some delay in the time or clearance of the right side of the heart. In infants and young children the concentration of contrast medium in the left heart is, on the other hand, generally satisfactory to produce a demonstrable shunt of the medium. Roentgenography in two projections simultaneously enables one to judge whether only the pulmonary artery remains opacified, whether it is revisualized separately or whether contrast medium at the same time is seen also in the right ventricle. By using a rapid sequence of films, it is further possible to register whether the concentration of the medium in the pulmonary artery diminishes successively in the normal way or whether the opacification is apparently prolonged, or if it even increases at all. Depending on the size of the shunt during the cardiac cycle, the reopacification is sometimes more evident in ventricular systole, sometimes in diastole (Fig. 5).

SELECTIVE ANGIOCARDIOGRAPHY

As heart catheterization is more and more commonly used, it was a logical development of the technique to inject the contrast medium through a catheter as

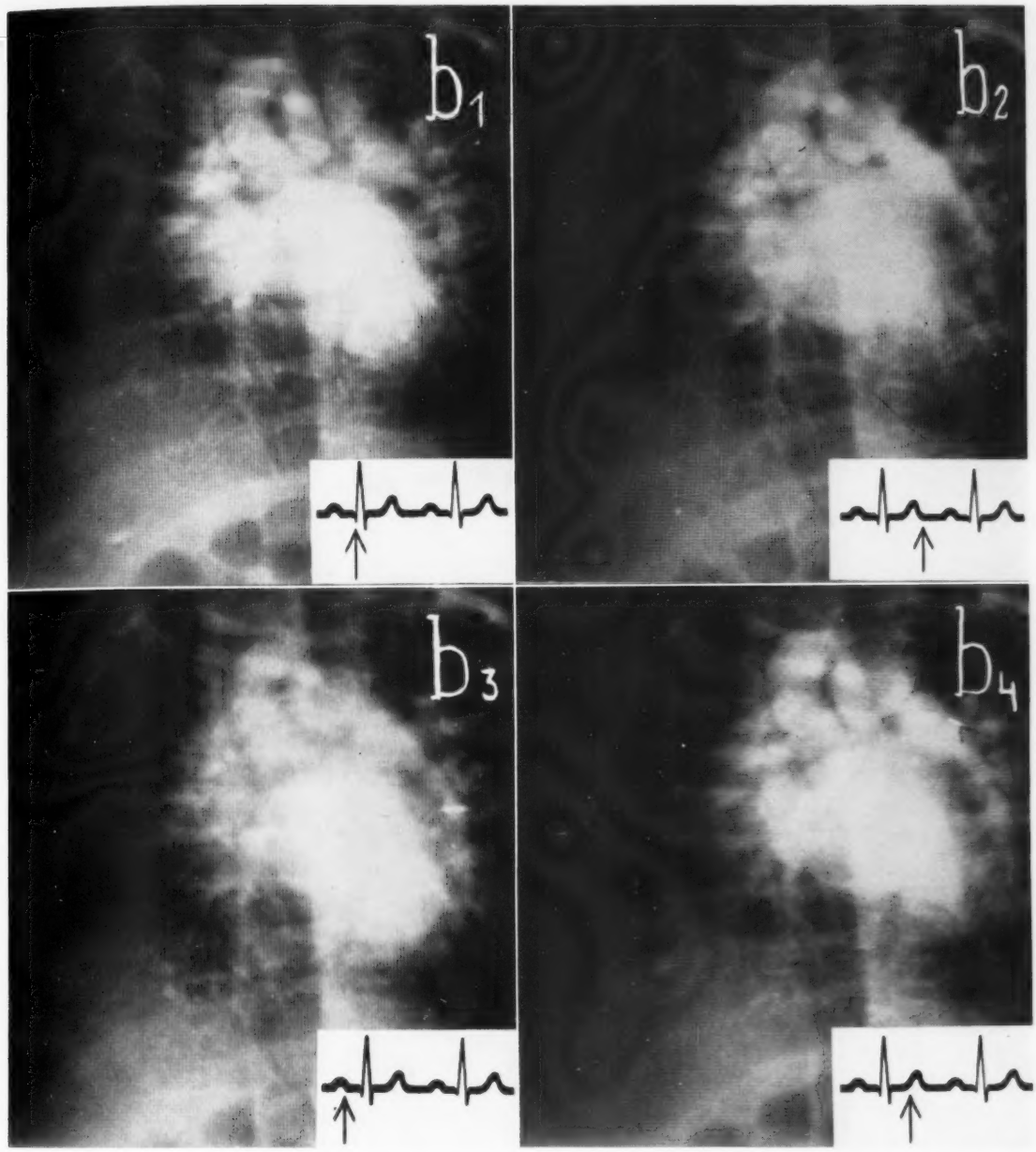

Fig. 5. *Systolic revisualization of the pulmonary artery in a case of patent ductus arteriosus.* Left anterior oblique projection.

(b_1) End of atrial systole. The left atrium, ventricle and aorta are contrast filled. Much medium is in the main branches and ramifications of the pulmonary artery, but no medium is seen in the main stem.

(b_2) End of the following ventricular systole. The left atrium is well filled. The ventricle is nearly empty. The main stem of the pulmonary artery is clearly seen and the concentration of the contrast medium has increased also in the branches of this vessel.

(b_3) Beginning of atrial systole. There is still contrast medium in the main stem of the pulmonary artery, but definitely less than in b_2.

(b_4) During the following ventricular systole the pulmonary artery reappears distinctly.

close to the area involved as possible (Jönsson, 1949). For the anatomic study much can be gained by not having other heart cavities or vessels simultaneously filled, thus perhaps projecting over and possibly masking the findings desired. The decision as to where the contrast medium should be injected is based on the results of the heart catheterization.

If the catheter passes through the ductus arteriosus, the contrast medium can be injected into the aorta with the tip of the catheter at the level of the entrance of the ductus arteriosus. In order to obtain optimal filling of the ductus, Kjellberg, *et al.* (1955) recommend that injection be made during withdrawal of the catheter from the aorta into the pulmonary artery.

In cases associated with a ventricular septal defect or a pulmonary valvular

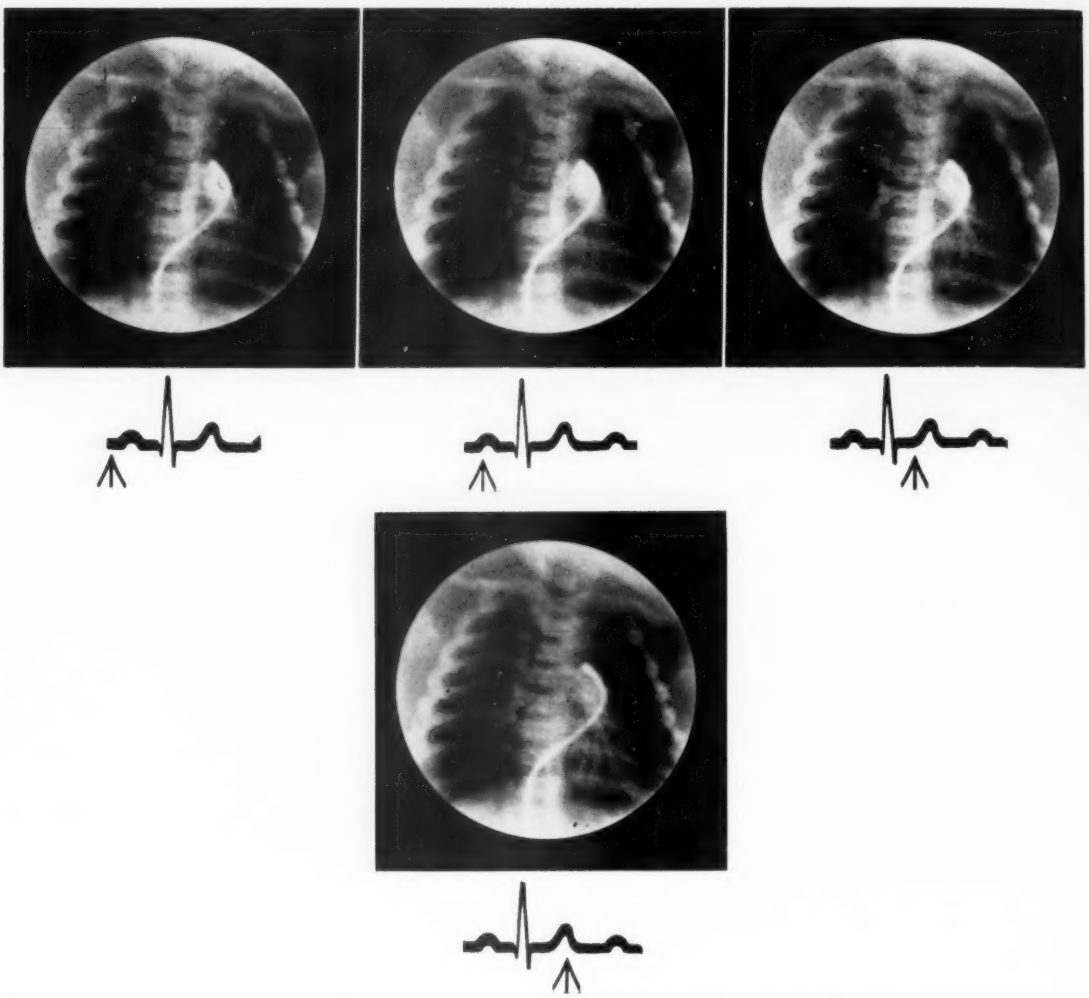

FIG. 6. *Patent ductus arteriosus.* Cineroentgenography with image intensification. Rate of exposures, 50 per second. (Selected pictures from a sequence, time related to the heart phase by electrocardiographic markings.) Anteroposterior projection. Contrast injection into the left pulmonary artery.

(b_1) Contrast filling of left pulmonary artery. Immediate passage of some contrast medium into the descending aorta.

(b_2) One fiftieth of a second later the contrast medium is more dense but has not proceeded either in the pulmonary artery or in the aorta.

(b_3) Synchronously with starting ventricular systole, the small branches of the pulmonary artery fill with opaque material which also is seen to have moved downwards into the aorta.

(b_4) Later in the same systole the small branches of the pulmonary artery are also seen.

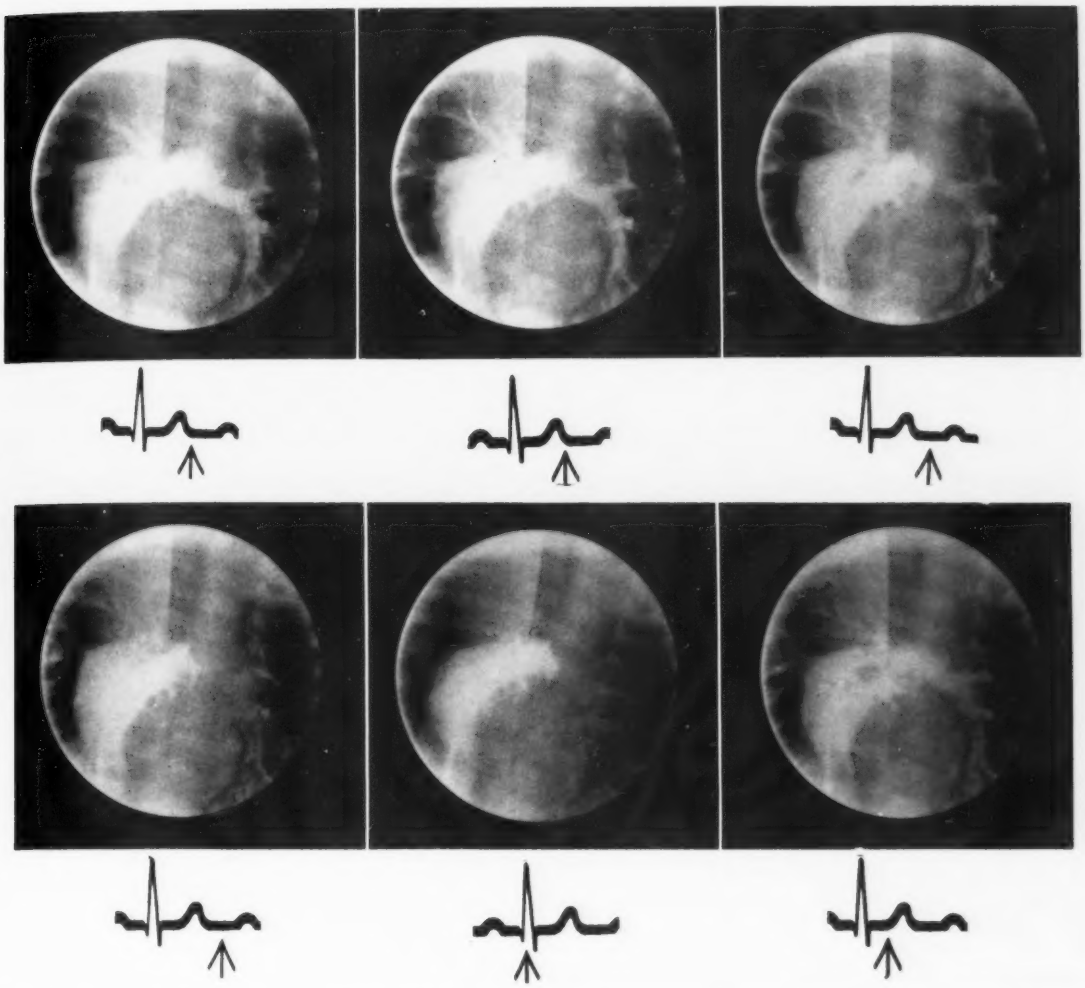

FIG. 7. *Patent ductus arteriosus. Left to right shunt.* Cineroentgenography with image intensification. Rate of exposures, 50 per second. Left anterior oblique projection. Contrast injection into the left pulmonary artery.

(b_1–b_6) Series of pictures showing the successively increasing diastolic dilution of the contrast medium in the pulmonary artery.

stenosis, the contrast medium may preferably be injected into the right ventricle with the purpose to outline the anatomy of the complicating malformation.

If the injection of contrast medium is made into the pulmonary artery, it will reach the left side of the heart less diluted than if the injection is made intravenously. The reopacification of the pulmonary artery is thus more strikingly demonstrated. On the other hand, the diastolic dilution of the contrast medium in the pulmonary artery appears less regularly, due to the disturbance of the hemodynamic conditions following the rapid injection. Sometimes, it is, therefore, possible to demonstrate the ductus arteriosus directly. A rapid injection of the contrast medium may cause a rise in pressure and reverse a left to right shunt during ventricular diastole. During ventricular systole, the direction of the shunt is again, however, from left to right. (Fig. 6). Conclusions concerning the direction of the shunt normally must, therefore, be drawn with great caution.

DISCUSSION

In view of the desirability, on the one hand, of being able to establish the diagnosis of patent ductus arteriosus at an early

stage for surgical consideration and, on the other hand, because of the technical difficulties in obtaining a satisfactory direct anatomic visualization of the patent ductus in infants, every additional aid is necessary when this malformation is suspected. Among roentgenologic facilities at our disposal, one may include intravenous fast angiocardiography with rapid sequence exposures which permits the addition of numerous valuable indirect signs of pathophysiologic nature to the currently used direct signs of anatomic visualization of this important malformation. By following such procedure it is possible to co-ordinate hemodynamic observations with morphologic data, thereby considerably enriching our possibilities in accurate roentgenologic diagnosis.

SUMMARY

After emphasizing the importance of an early diagnosis for surgical consideration in cases of patent ductus arteriosus, the angiocardiographic possibilities at our disposal are discussed. The indirect roentgenologic signs, mainly of a functional nature, play an important role in infants because of the technical difficulties in obtaining a direct visualization of the patent ductus arteriosus. When such indirect signs are present, a correct diagnosis may be established roentgenologically even in such instances in which the lesion cannot be visualized anatomically. The technique to be used demands a rapid exposure of three to four pairs of films synchronously in two planes per heart cycle, with simultaneous electrocardiographic registration and automatic exposure marking.

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ROENTGENOLOGIC ASPECTS OF THE EISENMENGER COMPLEX*

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THE Eisenmenger complex is one of the less common congenital heart malformations which may cause cyanosis. Although attempts at its surgical amelioration have been made,[23] it remains an inoperable condition, in contrast to some of the other congenital anomalies. In view of the increasing importance of exact diagnosis in congenital heart disease, it was felt that a careful roentgenologic analysis of a group of proved cases of the Eisenmenger complex might be rewarding. This paper will summarize existing pathologic and physiologic concepts, briefly describe the clinical data, and analyze the roentgenologic findings in the Eisenmenger complex, with emphasis on the roentgen appearance of the pulmonary vessels.

DEFINITION

Although Dalrymple[14] first described this anomaly in 1847 and Eisenmenger[18] reported and illustrated a case in detail in 1897, it was Abbott and Dawson[1] who emphasized the distinction between the Eisenmenger complex and uncomplicated defects of the membranous ventricular septum many years later. Both Eisenmenger and Abbott considered a ventricular septal defect, aortic overriding, a dilated pulmonary artery and deformed aortic cusps the main features of this malformation. Taussig,[32] elaborating on the clinical features in 1947, emphasized the presence of abnormalities in the pulmonary vessels.

Selzer and Laqueur[28] analyzed the literature and a number of personal cases in 1951. Their summary of modern concepts significantly broadened the definition. Thus, in discussing overriding of the aorta, they pointed out that this might be marked, minimal, or even absent; the important feature was the presence of arterial anoxemia as evidence that venous blood was entering the aorta. They stressed the importance of pulmonary hypertension in determining the direction of blood flow through a ventricular septal defect. In brief, they suggested that increased resistance to flow through the pulmonary circulation distinguishes the Eisenmenger complex from the uncomplicated ventricular septal defect, and that in all defects of the membranous septum the potentiality exists for right ventricular blood to enter the aorta directly. The realization of this potential depends on existing pressure relationships and the size of the defect, as well as on the degree of aortic overriding.

Thus, the definition of the Eisenmenger complex, as used in this paper, embodies both anatomic and physiologic components: a large ventricular septal defect with aortic overriding of variable degree, increased pulmonary vascular resistance and consequent pulmonary hypertension, and a shunt of right ventricular blood into the aorta.

METHODS AND MATERIAL

During the past four years we have performed roentgenologic studies on 12 patients with the Eisenmenger complex. All cases have had cardiac catheterization, with intracardiac and pulmonary arterial pressure measurements using a Hamilton manometer, and oxygen saturation studies. The physiologic criteria for diagnosis were the presence of pulmonary and right ventricular hypertension, absence of pulmonic stenosis, evidence of a ventricular septal defect, and demonstration of a right-to-left (veno-arterial) shunt of blood from right ventricle to aorta.[7,13,19,27]

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On all patients, fluoroscopic and roentgenographic studies were performed, including posteroanterior, right anterior oblique, left anterior oblique, and left lateral films. Kymography was occasionally performed, but added little significant information.

Over-all heart size, individual chamber size, main and branch pulmonary artery size and the aortic configuration were evaluated in a review of this material. Clinical and physiologic data were analyzed in conjunction with the roentgenologic material.

Clinical Data

The age of the patients varied from four to thirty-four years, with an equal sex distribution. In all cases cardiac or respiratory disease was suspected at birth or early in life because of cyanosis, poor growth, difficult breathing, or the detection of a heart murmur. Cyanosis was present in all but 1 case and was usually slight to moderate in degree. In 2 instances, cyanosis was noted at birth. Exertional dyspnea was a common complaint, and recurrent respiratory infections were present in two-thirds of the cases. Four patients had a history of frequent severe bouts of pneumonia. Hemoptysis, epistaxis, malaise, and impaired physical development were also occasionally present.

On physical examination, in addition to cyanosis, clubbing was noted in about one-half of the patients. All had murmurs, usually systolic, but occasionally diastolic as well, varying in location, intensity quality, and radiation. Although most commonly heard in the fourth intercostal space at the left sternal border, the murmurs were also heard at the apex and the base, and were in no way characteristic. Occasionally a classical Graham-Steell murmur was present. A thrill was rarely palpable. The second pulmonic sound was accentuated in all cases and described as "snapping" in some. In half of the cases, the chest cage was deformed, with marked forward protrusion of the left hemithorax due to right ventricular enlargement.

A review of the literature reveals relatively little variance from the clinical findings in this group of patients.[8,9,13,19,20] It is of interest that a history of multiple pulmonary infections, which is prominent in our cases, has not previously been stressed. The absence of a "characteristic" murmur conforms to other reports. Similarly, the generally mild to moderate degree of cyanosis is well established, although cyanosis may be present at birth,[28] as noted in some of our patients.

Laboratory Data

Slight elevation of hematocrit and red blood cell count was commonly found. The electrocardiogram showed a right ventricular hypertrophy pattern in 11 instances and right bundle branch block in 1. P-wave changes were noted in 4 cases consisting primarily of peaked, Gothic-type waves in leads 2 or 3. Except for a slightly higher incidence of P-wave alterations in our cases, the electrocardiographic findings are in agreement with those previously reported.

Physiologic Data

Marked elevation of right ventricular and pulmonary artery pressure, with equalization of these pressures and the systemic pressure, was present in all instances. Although some patients had a small net left-to-right shunt and others a small net right-to-left shunt, the usual finding was one of small, mixed, bi-directional shunts. In all cases the presence of a ventricular septal defect was demonstrated either by catheterization of the defect or by increased oxygen saturation in the right ventricle as compared to the right atrium. All cases had arterial anoxemia of varying degree. The average arterial oxygen saturation was 84 per cent.

Roentgenologic Findings

1. *Heart Size.* The over-all heart size in the posteroanterior projection was not markedly increased (Fig. 1 and 2). One-half of the patients had no increase in transverse cardiac diameter, and the remainder,

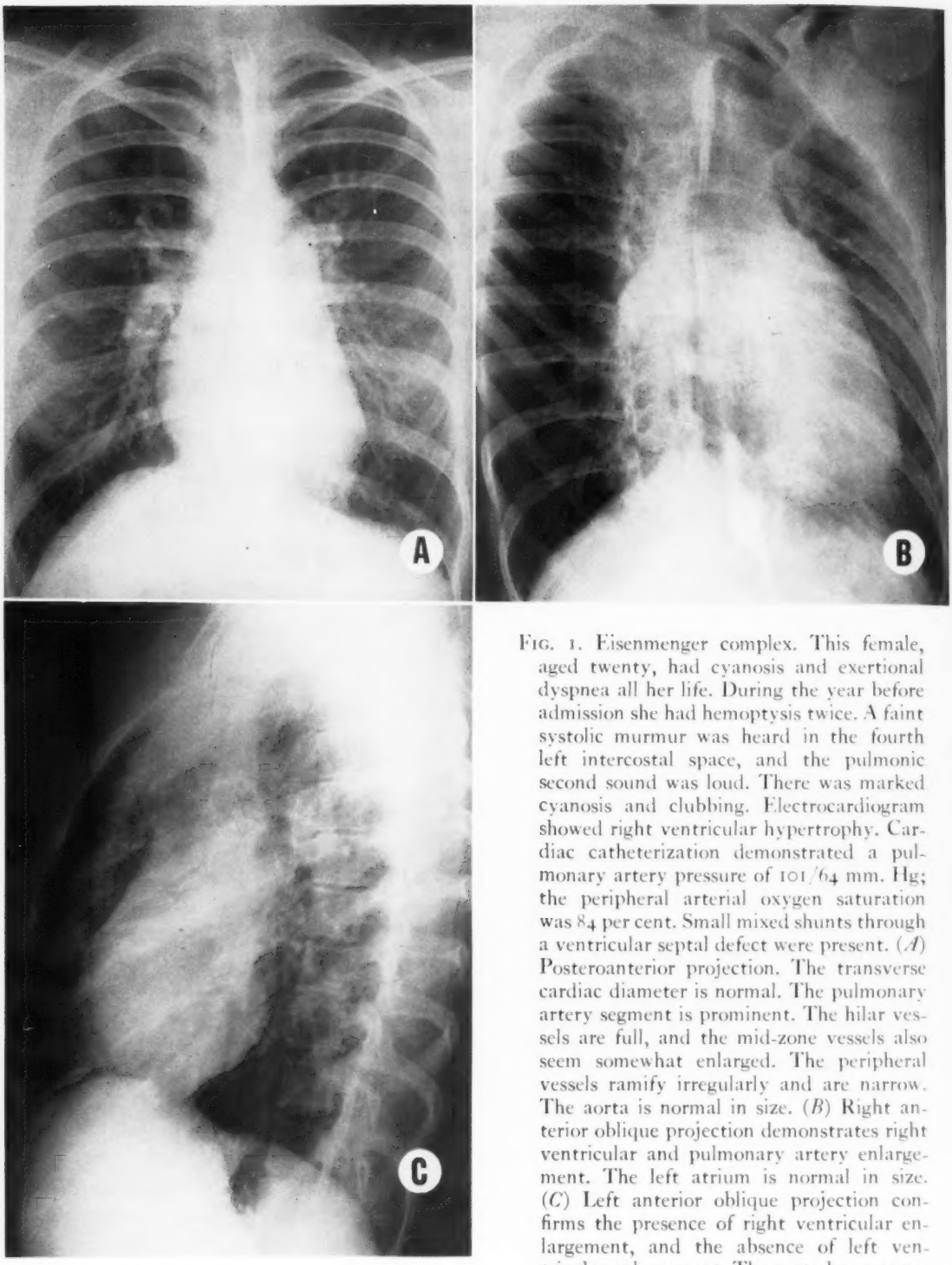

FIG. 1. Eisenmenger complex. This female, aged twenty, had cyanosis and exertional dyspnea all her life. During the year before admission she had hemoptysis twice. A faint systolic murmur was heard in the fourth left intercostal space, and the pulmonic second sound was loud. There was marked cyanosis and clubbing. Electrocardiogram showed right ventricular hypertrophy. Cardiac catheterization demonstrated a pulmonary artery pressure of 101/64 mm. Hg; the peripheral arterial oxygen saturation was 84 per cent. Small mixed shunts through a ventricular septal defect were present. (*A*) Posteroanterior projection. The transverse cardiac diameter is normal. The pulmonary artery segment is prominent. The hilar vessels are full, and the mid-zone vessels also seem somewhat enlarged. The peripheral vessels ramify irregularly and are narrow. The aorta is normal in size. (*B*) Right anterior oblique projection demonstrates right ventricular and pulmonary artery enlargement. The left atrium is normal in size. (*C*) Left anterior oblique projection confirms the presence of right ventricular enlargement, and the absence of left ventricular enlargement. The aorta has a somewhat anterior takeoff, suggesting the possibility of overriding.

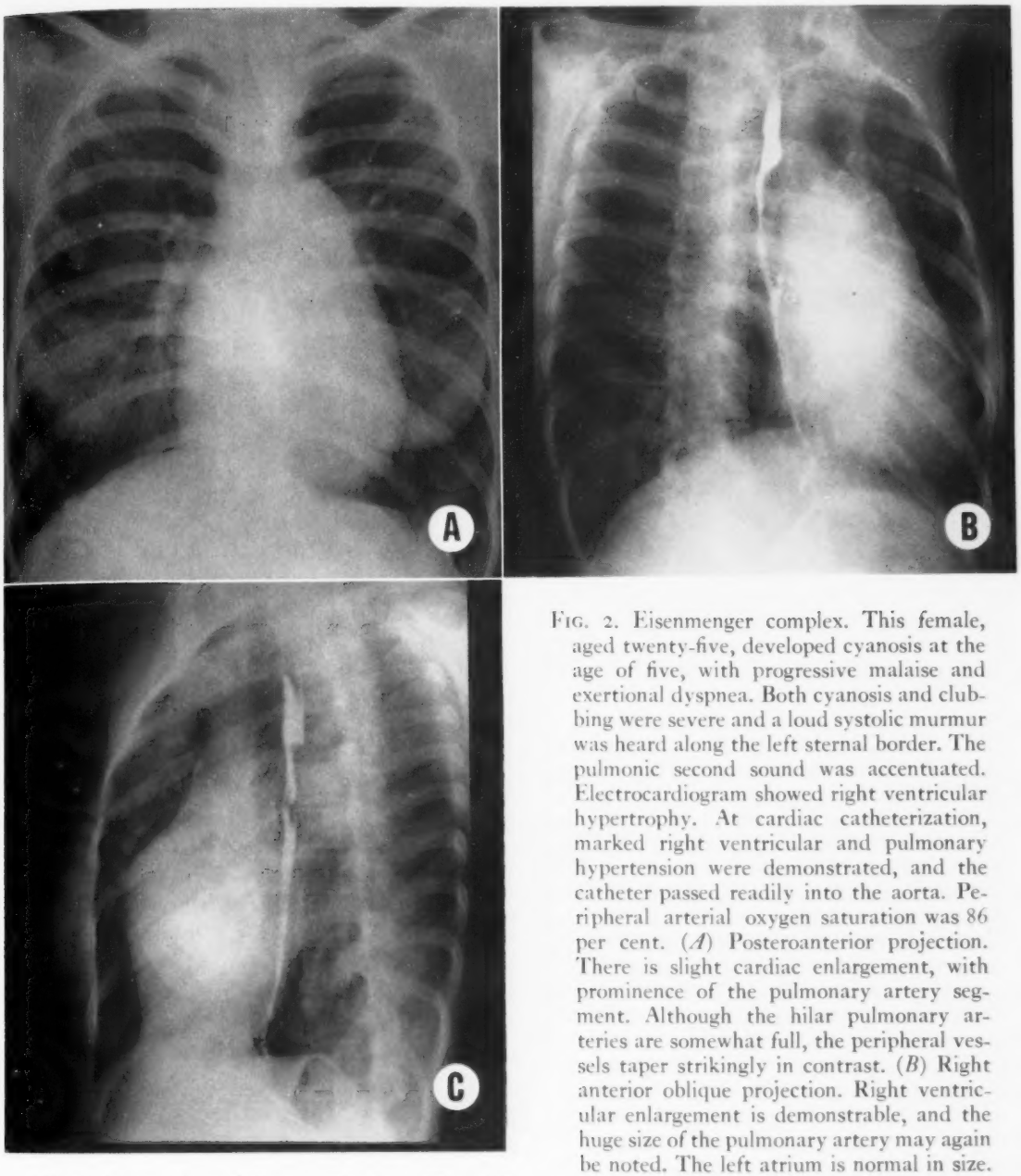

Fig. 2. Eisenmenger complex. This female, aged twenty-five, developed cyanosis at the age of five, with progressive malaise and exertional dyspnea. Both cyanosis and clubbing were severe and a loud systolic murmur was heard along the left sternal border. The pulmonic second sound was accentuated. Electrocardiogram showed right ventricular hypertrophy. At cardiac catheterization, marked right ventricular and pulmonary hypertension were demonstrated, and the catheter passed readily into the aorta. Peripheral arterial oxygen saturation was 86 per cent. (*A*) Posteroanterior projection. There is slight cardiac enlargement, with prominence of the pulmonary artery segment. Although the hilar pulmonary arteries are somewhat full, the peripheral vessels taper strikingly in contrast. (*B*) Right anterior oblique projection. Right ventricular enlargement is demonstrable, and the huge size of the pulmonary artery may again be noted. The left atrium is normal in size. (*C*) Left anterior oblique projection. The right ventricle projects anteriorly, and the segment formed by the right atrial appendage seems somewhat lengthened, suggesting right atrial enlargement. There is no evidence of left ventricular enlargement.

with one exception, had only slight to moderate enlargement. In 1 case (Fig. 3), there was evidence of marked bi-ventricular dilatation; this patient had the greatest systemic and pulmonary blood flow. No other correlation between over-all heart size and pulmonary or systemic flow, pulmonary artery pressure or pulmonary vascular resistance was definable.

a. Right ventricular enlargement, as evaluated in all 4 projections, was present to some degree in all cases, although in only 3 cases was it marked. No specific correlation with the physiologic data could be es-

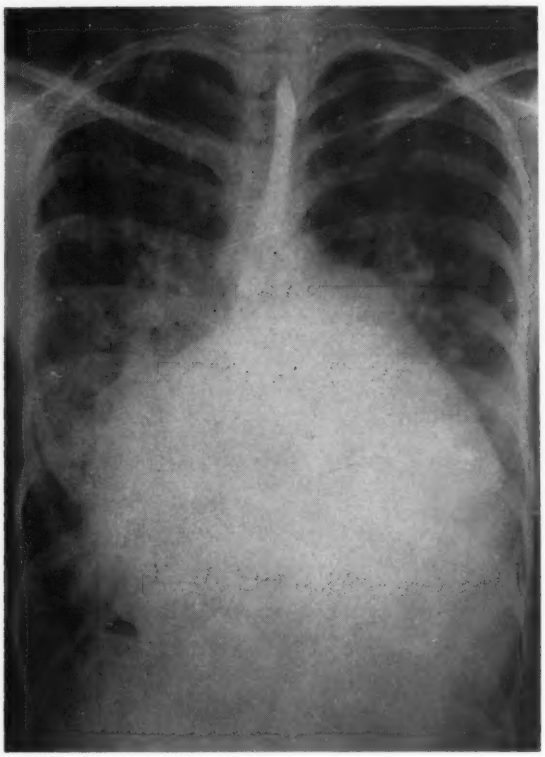

FIG. 3. Eisenmenger complex. This female, aged twenty-four, had a heart murmur since early childhood, and developed congestive heart failure one year before admission. She had massive cardiac enlargement, and a systolic murmur and thrill. The pulmonic second sound was loud. Electrocardiogram showed incomplete right bundle branch block. On cardiac catheterization, marked pulmonary hypertension and a ventricular septal defect with large mixed shunts were present. Arterial oxygen saturation was 86 per cent. Posteroanterior roentgenogram shows massive cardiac enlargement, a huge pulmonary artery segment, and large hilar branches. The mid-zone and peripheral arteries are slightly more prominent than usual in the Eisenmenger complex. Note the presence of "Kerley's lines" at the right costophrenic sulcus. These are probably more an indication of venous or lymphatic engorgement than they are of pulmonary hypertension *per se*.

tablished. It was of interest that the 3 cases with the greatest right ventricular enlargement had the lowest pulmonary artery and right ventricular pressures in the group.

b. Left ventricular enlargement, best judged in the posteroanterior and left anterior oblique projections, was not a striking feature in these cases. Slight enlargement was noted in 3 cases, and moderate enlargement in 2.

c. Right atrial size was best evaluated in the posteroanterior, right anterior oblique, and left anterior oblique projections. In two-thirds of the cases, there was suggestive evidence of right atrial enlargement.

d. Left atrial size, as determined in the posteroanterior and the right anterior oblique projections, was normal in all but 1 case, in which it was equivocally enlarged.

e. Changes in heart size. In 2 of our cases, followed over a period of four years (Figs. 4 and 5), there was a definite decrease in over-all heart size with increasing age. This seemed greater than the normal age change in thoracic configuration alone could explain, particularly in 1 instance (Fig. 5). In this case, massive cardiac enlargement at the age of eight months had significantly diminished by the age of four years, and has since decreased even further. We have previously noted massive cardiac enlargement and congestive heart failure in septal defects with huge left-to-right shunts in infancy.[3] The development of increased vascular resistance in these cases as a means of diminishing flooding of the lungs, and the shift from a massive left-to-right shunt to small bi-directional shunts may well be accompanied by a distinct diminution in heart size.

2. *The Pulmonary Artery and Its Branches.* Evaluation of the caliber of the pulmonary arterial vessels was divided into 4 separate phases:[2] (a) the main pulmonary artery, the size of which was judged by the relative prominence of the pulmonary artery segment of the left heart border in the posteroanterior and right anterior oblique projections; (b) the hilar or central branches of the pulmonary artery, represented by the caliber of the right descending pulmonary artery at its widest points; (c) the mid-zone branches of the right lung; and (d) the peripheral branches (Fig. 6).

a. The main pulmonary artery was enlarged in all instances and grossly dilated

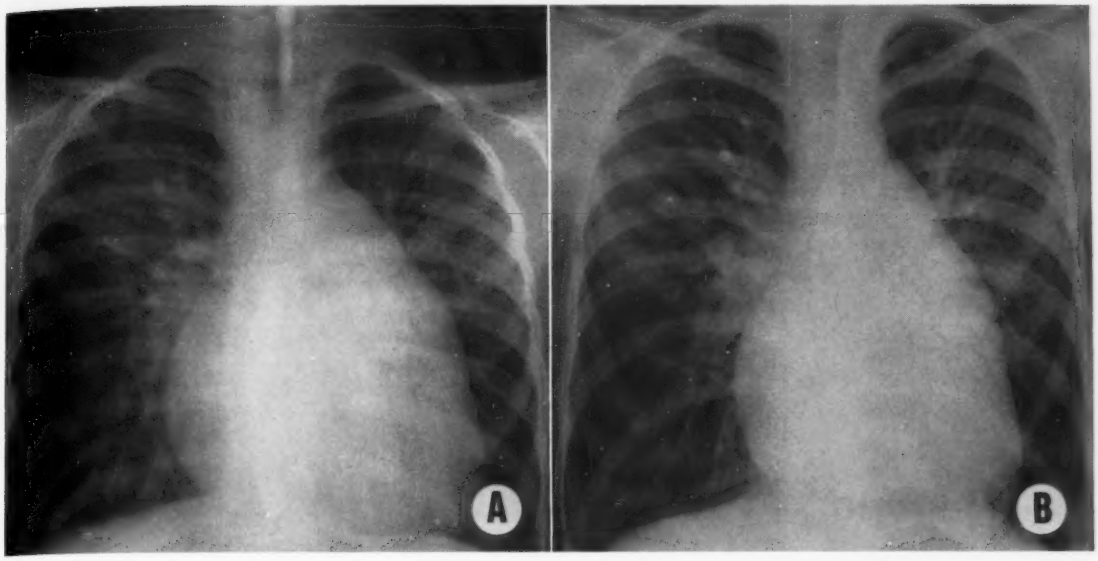

Fig. 4. Eisenmenger complex. This male, aged thirteen, had a heart murmur and cardiac enlargement at birth. He suffered frequent bouts of respiratory infection. There was slight cyanosis but no clubbing. An apical systolic and diastolic murmur was heard. The pulmonic second sound was loud and split. Electrocardiogram showed right ventricular hypertrophy. Marked pulmonary hypertension and a ventricular septal defect were found on catheter study. Femoral artery oxygen saturation was 88 per cent. (*A*) Posteroanterior projection at the age of nine years. Gross cardiac enlargement is present, and there is prominence of the pulmonary artery segment. The left atrial appendage projects somewhat more than usual, but no evidence of left atrial enlargement was present in the oblique views. The hilar pulmonary arteries were enlarged, and the mid-lung branches were also somewhat prominent. The peripheral vessels, however, were small and delicate. (*B*) Posteroanterior projection at the age of thirteen. The marked cardiac enlargement previously observed is no longer present, although the contours of the heart have not changed significantly. The disparity between the size of the central and the peripheral vessels is slightly more marked at this time than previously. The aorta appears hypoplastic.

in 8 of 12 cases. Its size was usually assessed with ease in the posteroanterior projection, although in 1 case the right anterior oblique projection demonstrated massive enlargement not as well seen in the frontal plane. In a few instances, aneurysmal dilatation of the main pulmonary artery was apparent (Fig. 7).

b. The central pulmonary arteries were also consistently enlarged (Fig. 1, 3, 5 and 7), markedly so in two-thirds of the cases. Vigorous pulsation of the main and hilar pulmonary arteries was observed at fluoroscopy in most instances.

c. The mid-zone branches were more prominent than normal in most cases, but only slightly so in some instances. On comparison with cases in which large left-to-right shunts were present without pulmonary hypertension, the mid-zone branches in the Eisenmenger complex were less enlarged than those associated with hilar branches of equivalent size in the left-to-right shunts.

d. Peripheral branches in all but 1 case were disproportionately small in relation to the caliber of the enlarged hilar vessels (Fig. 1, 2, 4, 7 and 8). In 6 cases, the peripheral vessels were distinctly attenuated or at the lower limits of normal size. The vessels appeared normal in 3 cases and slightly prominent in 2, but in all of these the ratio of the size of the central vessel to that of the peripheral vessels was increased.

3. *The Aorta.* In three-fourths of the cases, the aorta appeared hypoplastic (Fig. 2, 4, 5 and 7). This was true in spite of the fact that systemic blood flow was not re-

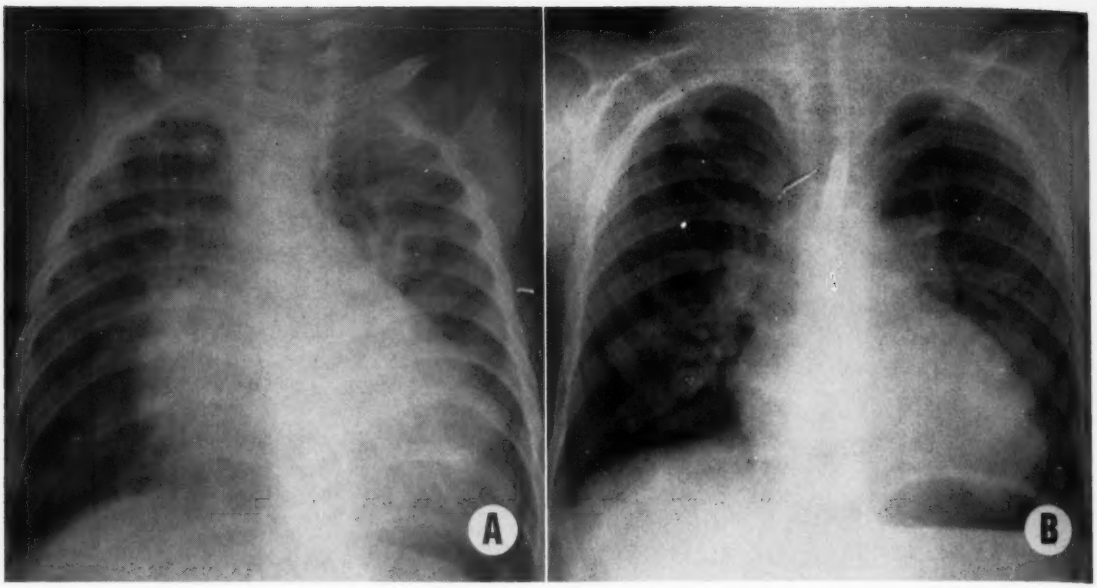

FIG. 5. Eisenmenger complex. This male, aged four, was first seen at the age of eight months because of poor growth, congestive heart failure, and slight cyanosis. He had gross cardiac enlargement. A systolic murmur was heard along the left sternal border, and the pulmonic second sound was loud. Electrocardiogram showed right bundle branch block. He was catheterized at the age of four years, at which time the pulmonary artery pressure was 91/29 mm. Hg; small mixed shunts through a ventricular septal defect and a peripheral oxygen saturation of 79 per cent were also demonstrated. (*A*) Posteroanterior projection at 8 months of age. There is massive cardiac enlargement, which in the oblique projections involved both the right and left ventricles. The hilar pulmonary arteries are full, but there is not as much peripheral engorgement as might be expected in the presence of a large left-to-right shunt. The aorta is not well visualized. (*B*) Posteroanterior, projection at 4 years of age. The heart has diminished in relative size during the interval. The central pulmonary arteries are quite full, and the mid-lung branches are moderately enlarged. The peripheral arteries, however, are of small caliber. The oblique projections showed evidence of marked right ventricular enlargement, and some left ventricular enlargement as well.

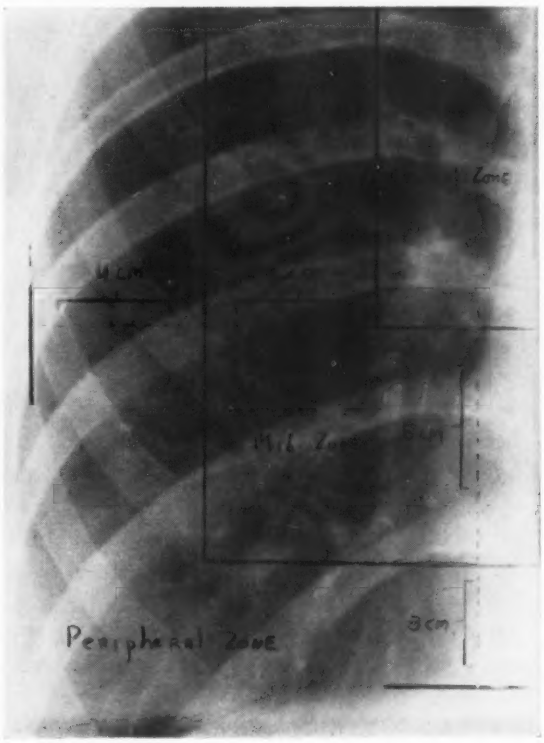

FIG. 6. Evaluation of the size of the pulmonary artery branches. The lower two-thirds of the right lung field were used as the basis for evaluating the size of the pulmonary branches. The central (or hilar) pulmonary artery size was considered equivalent to the size of the right descending pulmonary artery at its widest point, and a "central zone" was established, the lower border of which corresponded to the origin of the right middle lobe artery, and the lateral border was the lateral border of the right descending pulmonary artery. A "mid-zone" was arbitrarily designated as the upper two-thirds of the area between the lower border of the central zone and the diaphragm. The distal one-third was considered the peripheral zone. The lateral margin of the mid-zone was fixed at a point half-way between the margin of the central zone and the rib cage. These divisions allow relatively reproducible sites for inspection and measurement of vessel size, and comparisons may then be made with normal cases. All roentgenograms were obtained in inspiration.

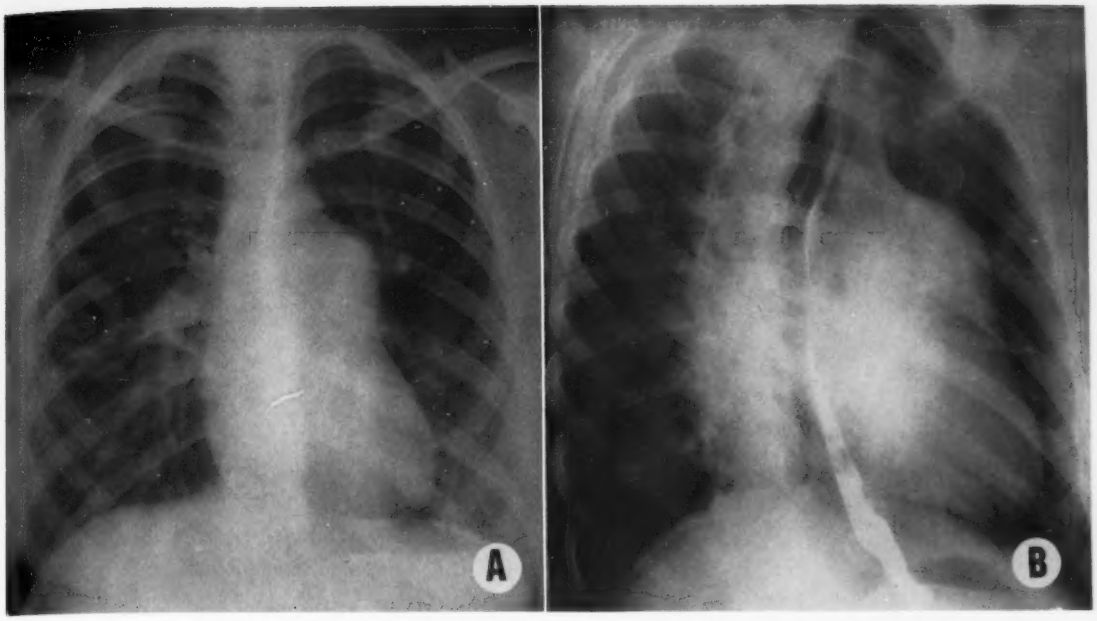

FIG. 7. Eisenmenger complex. This female, aged seventeen, was first discovered to have a heart murmur at three years of age. She was mildly cyanotic; systolic and diastolic murmurs were heard to the left of the precordium, with a loud pulmonic second sound. Electrocardiogram showed right ventricular hypertrophy. Cardiac catheterization showed a pulmonary artery pressure of 93/56 mm. Hg, with small mixed shunts through a ventricular septal defect. Peripheral arterial oxygen saturation was 86 per cent. (*A*) Posteroanterior projection. The transverse cardiac diameter is normal. The main pulmonary artery is massively enlarged and projects prominently to the left. The hilar pulmonary arteries are also enlarged. The arterial branches in the mid lung are moderate in size, but there is a striking reduction in caliber of the peripheral vessels. This is the classical appearance of pulmonary hypertension. (*B*) Right anterior oblique view. Note the huge aneurysmal bulge of the main pulmonary artery. The barium-filled esophagus demonstrates no evidence of left atrial enlargement. The right ventricle projects forward somewhat more than usual, indicative of some enlargement.

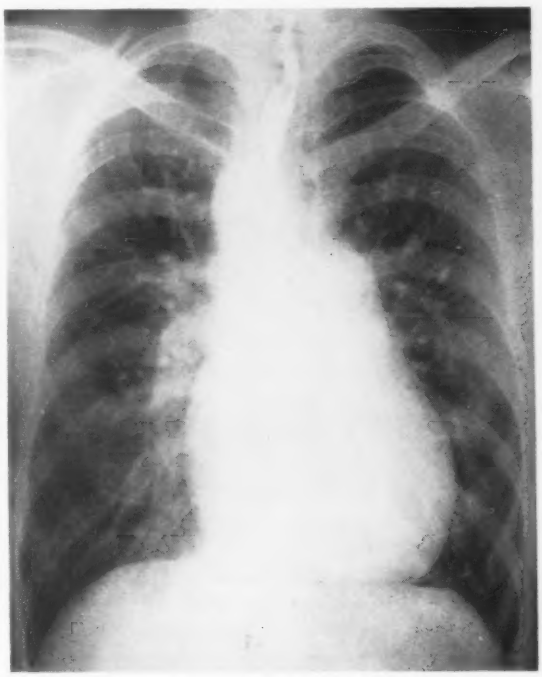

FIG. 8. Eisenmenger complex. This male, aged thirty-one, was cyanotic at birth, and had progressively increasing exertional dyspnea. Marked cyanosis and clubbing were present. The left chest bulged anteriorly. A long diastolic murmur was heard to the left of the sternum and the pulmonary second sound was loud. Electrocardiogram showed right ventricular hypertrophy. The pulmonary artery pressure of 108/64 mm. Hg exceeded the aortic pressure (101/58 mm. Hg), and the aorta was directly catheterized from the right ventricle. Peripheral arterial oxygen saturation was 83 per cent at rest and 63 per cent on exercise. The posteroanterior projection demonstrates slight cardiac enlargement, and a prominent pulmonary artery segment. The aorta is right-sided and indents the esophagus from the right. The hilar pulmonary arteries, best seen on the right, are full, but both the mid-lung and the peripheral arteries taper more sharply than might be expected from the size of the central vessels.

duced. Since a small aorta is not an anatomic characteristic of the Eisenmenger complex, it seems possible that enlargement of the main pulmonary artery may be sufficiently marked to obscure the aortic knob and make the aorta seem smaller than it really is. In 1 case a right aortic arch was present (Fig. 8). In 2, the aorta was somewhat anteriorly placed in the left anterior oblique projection, suggesting the presence of overriding.

A review of the literature indicates that the roentgenologic findings in the Eisenmenger complex have received relatively little attention. Taussig[32] noted that enlargement of the main pulmonary artery need not occur and that confusion with the tetralogy of Fallot might arise under these circumstances. In our experience, it is only in infancy or early childhood that the pulmonary artery is not markedly enlarged. Certainly in adult life, the absence of definite enlargement of the main pulmonary artery militates against the diagnosis of the Eisenmenger complex. Selzer, in a review of 35 autopsied cases, found a "remarkable uniformity in the roentgenographic appearance." He stated that the "pulmonary artery shadow and that of its branches were usually described as prominently enlarged, often extreme in degree. Prominent hilar pulsations were usually noted. . . . The heart was almost always enlarged."[27] Although others[8,13] have commented on the roentgenologic features, the importance of careful analysis of the caliber of the pulmonary vessels, both centrally and peripherally, has not been adequately stressed. A more recent discussion[34] of pulmonary hypertension has emphasized the relatively small size of the peripheral vessels by contrast with the large central arteries.

DISCUSSION

The conventional roentgenologic silhouette found in the Eisenmenger complex usually exhibits a striking similarity from case to case. Used in conjunction with the clinical data, a specific diagnosis can frequently be made and may be corroborated at cardiac catheterization. Prior to physiologic studies, a number of lesions must, however, be considered in the differential diagnosis. Even following cardiac catheterization, the diagnosis may not be established if the catheter fails to enter the pulmonary artery, the septal defect, or the aorta. Thus, such anomalies as atrial septal defect or patent ductus arteriosus with pulmonary hypertension may require further study.

1. *The Role of Angiocardiography in Diagnosis.* Angiocardiography may be helpful in infants and children or in adults when the diagnosis is in doubt. The pattern may be quite typical. Slight to moderate enlargement of the right atrium and right ventricle is usually observed (Fig. 9, *A* and *B*). The outlet of the right ventricle demonstrates no area of persistent narrowing either in the infundibular region or at the valve. Simultaneously with filling of the pulmonary artery, aortic opacification occurs (Fig. 9, *C* and *D*). Usually, but not invariably, the aorta is less densely opacified than the pulmonary artery. The main pulmonary artery is always greatly dilated, as are its central branches. This dilatation extends into the midlung fields, but the vessels usually narrow significantly as they approach the periphery.

Premature aortic opacification establishes the presence of functional or anatomic overriding, and thus excludes some of the other anomalies usually considered in the differential diagnosis of the Eisenmenger complex. It permits conclusive differentiation from primary pulmonary hypertension. Since both the ascending and the descending aorta are opacified, patent ductus arteriosus with reverse flow may be ruled out. The absence of early left atrial opacification excludes atrial septal defect with pulmonary hypertension. That valvular and infundibular pulmonic stenosis are absent can be stated with reasonable certainty if studies are performed in both oblique projections, since the infundibulum and valve area are then well visualized.

One reservation must be stated. Failure

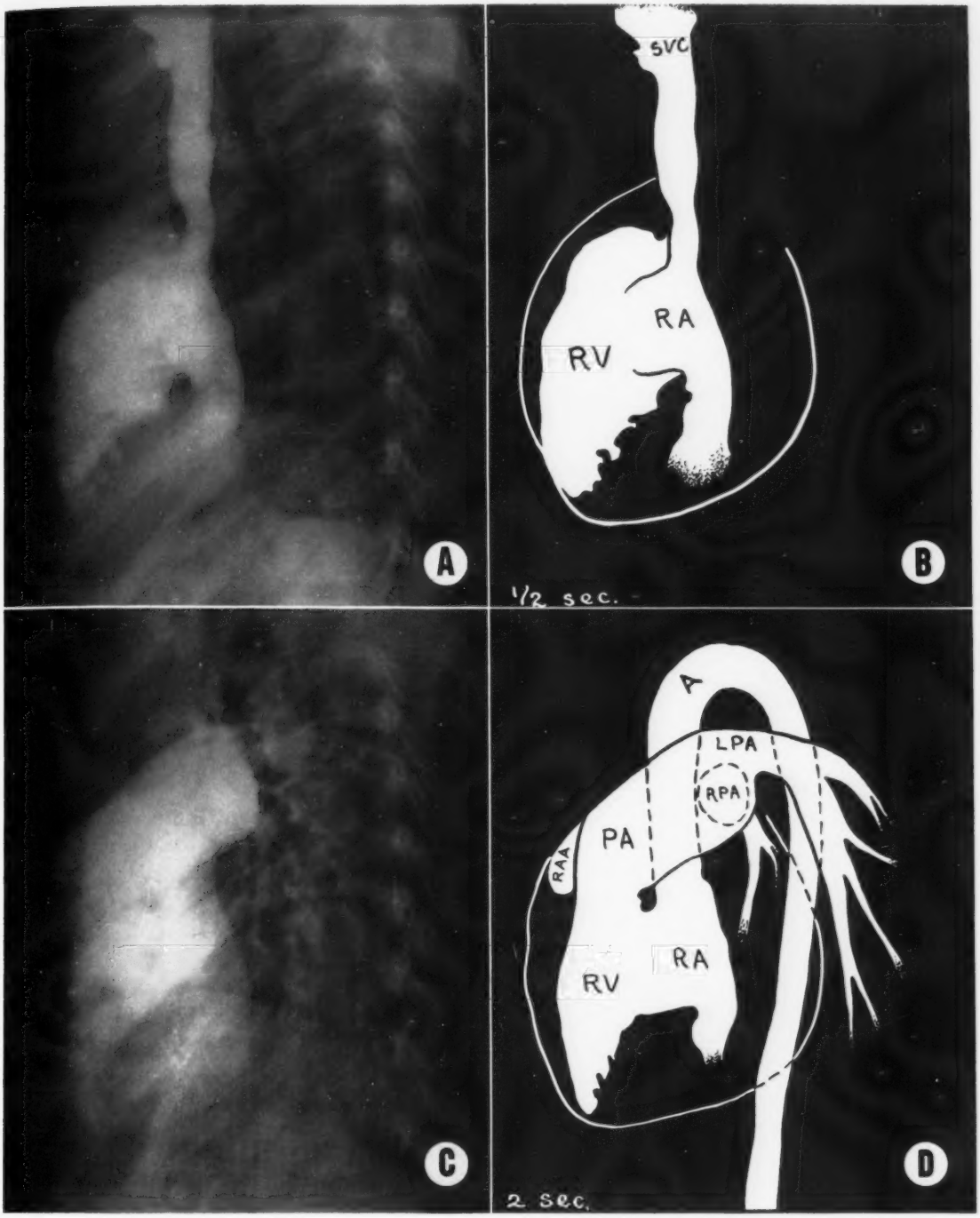

FIG. 9. Angiocardiogram in a female, aged seven, with the Eisenmenger complex. Right posterior oblique projection. (*A* and *B*) At 0.5 second, there is dense opacification of the superior vena cava, right atrium, and right ventricle, with some reflux into the inferior vena cava. (*C* and *D*) At 2 seconds, the pulmonary artery is densely opacified, simultaneously with aortic opacification, indicating the presence of aortic overriding with a shunt from the right ventricle, since the left ventricle is not as yet opacified. The pulmonary artery trunk and the main branches are grossly dilated, with a disproportionate diminution in the size of the peripheral branches, characteristic of pulmonary hypertension. It should be stressed that the presence of pulmonic stenosis cannot be excluded from the angiocardiogram alone in the right posterior oblique projection; pulmonary artery pressures must be ascertained by catheterization in order to establish a conclusive diagnosis. If the procedure is performed in the left posterior oblique projection (or the right anterior oblique projection), however, the outflow tract of the right ventricle and the pulmonary valve area are thrown into profile, and the presence of pulmonic stenosis can usually be detected with ease.

to demonstrate the presence of overriding angiocardiographically does not necessarily exclude it. The demonstration of overriding depends on the amount of right ventricular blood shunted in the aorta; if this is relatively slight, an unequivocal degree of opacification may not occur.

2. *The Diagnosis of Pulmonary Hypertension.* At present, the most useful technique for evaluating the presence and degree of pulmonary hypertension is cardiac catheterization. A less complicated approach might be of great value in the study of the cardiac patient. The clinical symptomatology may be suggestive of pulmonary hypertension, but is obviously not specific enough to rely on. The physical findings, such as an increased or split pulmonic second sound or right ventricular heave, may also indicate the presence of pulmonary hypertension, but are rarely conclusive. The electrocardiogram, by demonstrating right ventricular hypertrophy or right bundle branch block, is often helpful; obviously, however, identical findings might be expected in pulmonic stenosis.

Recently, attention has been focused on the possibility of conventional roentgenologic diagnosis of pulmonary hypertension.[2] Healey and his co-workers[20] made an important contribution in analyzing the effects of pulmonary hemodynamics on the roentgenologic appearance of the pulmonary arteries. A pulmonary blood flow greater than 7 l./min./m.^{2} caused an increase in the size of the pulmonary arteries, as did increased pulmonary arterial pressure. They could not distinguish the effects of "increased flow with normal pressure from increased pressure with normal flow." Campbell[10] confirmed their finding two years later, and pointed out that larger main pulmonary arteries were seen in the Eisenmenger complex, with increased pulmonary pressure and normal flow, than in the left-to-right shunts with normal pressure and markedly elevated flow.

Perhaps the most thorough roentgenologic studies of pulmonary hypertension have been performed in patients with mitral stenosis.[6,11,15,30,33] In general these studies reveal no correlation between the degree of cardiomegaly and the extent of pulmonary hypertension. Right ventricular enlargement correlated well with the presence but not with the degree of pulmonary hypertension. There was no relationship between left atrial size and the degree of pulmonary hypertension. Finally, an analysis of pulmonary artery size demonstrated at best a rough correlation between the size of the central pulmonary arteries and the degree of pulmonary hypertension in mitral stenosis; prominence of the pulmonary artery segment alone was of no value in predicting the presence or degree of pulmonary hypertension.

Angiocardiographic studies in mitral stenosis demonstrated varying degrees of occlusive change in the smaller, peripheral pulmonary arteries, while the main pulmonary artery and the central pulmonary arteries were dilated and large.[4,30] The degree of irregular narrowing and attenuation of the peripheral vessels was roughly proportional to the height of pulmonary arterial pressure as ascertained by catheterization. Careful study of the conventional roentgenograms in a large group of cases revealed that the narrow, irregular, tortuous distal arteries could usually be identified, and were well seen in the lower lobes. Others[11,33] have stressed the presence of so-called Kerley's lines in pulmonary hypertension. More recently, the association of pulmonary hypertension in congenital heart disease with large central and relatively narrow peripheral vessels has also been emphasized.[2,34]

The cases reported in this paper illustrate the roentgenologic pattern of severe pulmonary hypertension in congenital heart disease. The main and hilar pulmonary arteries are enlarged, while the peripheral branches are either normal in size or small. In patients with large left-to-right shunts and normal pressure, the main and hilar arteries are also enlarged, but the peripheral arteries show a commensurate increase in size (Fig. 10). Furthermore, in such pa-

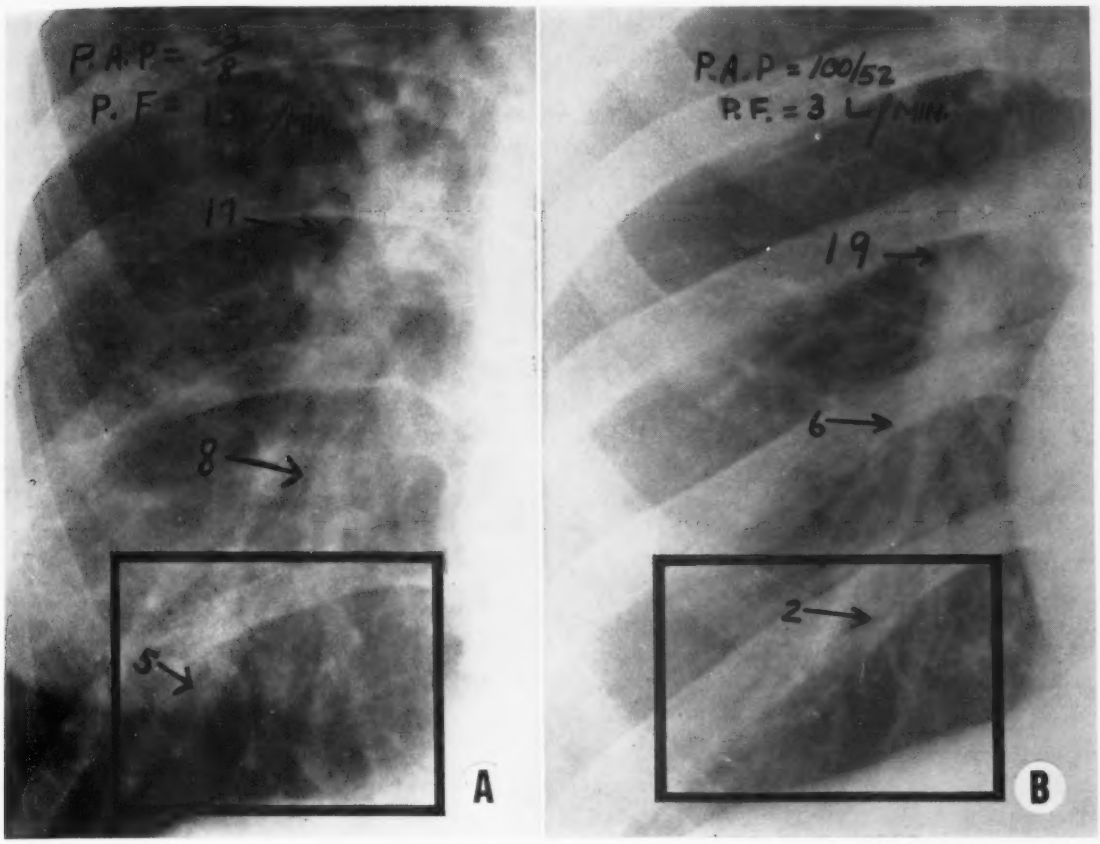

Fig. 10. Increased pulmonary flow versus increased pulmonary artery pressure. (*A*) *Increased flow.* The pulmonary artery pressure (PAP) is normal (23/8 mm. Hg), but the flow (PF) is 13 liters per minute because of the left-to-right shunt through an atrial septal defect. The central (17 mm.), mid (8 mm.), and peripheral (5 mm.) branches are all grossly but proportionately enlarged. Thus, there is no discernible discrepancy between the size of the central branches and that of the peripheral branches. (*B*) *Increased pressure.* The pulmonary artery pressure is markedly elevated (100/52 mm. Hg), but the pulmonary flow is decreased. Although the central pulmonary artery is grossly enlarged (19 mm.), the peripheral branches (2 mm.) taper strikingly and are uniformly small. This is the characteristic roentgenologic appearance associated with marked elevation of the pulmonary arterial pressure.

tients large pulmonary veins can frequently be visualized. Finally, in valvular pulmonic stenosis the main pulmonary artery may also be strikingly prominent, but in our experience post-stenotic dilatation never involves the right descending pulmonary artery.

We have compared the roentgen appearance in mitral stenosis complicated by pulmonary hypertension with that in our cases of the Eisenmenger complex.[2] Although the pulmonary arterial patterns were similar in many instances, Kerley's lines, present in many cases of mitral stenosis with severe pulmonary hypertension, were found in only 1 case of pulmonary hypertension in the Eisenmenger complex (Fig. 3). Kerley,[29] in describing these fine, horizontal, linear densities, best seen in the costophrenic angle, considered them evidence of lymphatic engorgement. Others[11] have related their presence to pulmonary hypertension. Since they are most commonly found in pulmonary hypertension associated with increased pulmonary venous pressure (as in mitral stenosis), and are rarely present when pulmonary arterial pressure is high but pulmonary venous pressure is normal, they cannot be considered a sign of pulmonary arterial hypertension *per se*. A

further roentgenographic characteristic of pulmonary hypertension in mitral stenosis is the presence of an increased number of peripheral pulmonary vessels.[33] This finding is not often observed in pulmonary hypertension associated with congenital heart disease.

The explanation of the increased central vessel size in the presence of decreased or normal peripheral vessel size in pulmonary hypertension can be briefly summarized. The central and at times the mid vessels enlarge because of the increased resistance in the pulmonary vascular bed. The peripheral vessels are relatively small because they may be the seat of medial thickening, secondary intimal thickening, or spasm on a neurogenic basis.[12,17,24,31]

3. *Differential Diagnosis.* The Eisenmenger complex must be differentiated from all conditions associated with pulmonary hypertension. The role of angiocardiography in this differentiation has already been discussed.

a. Atrial septal defect. Roentgenologically, an atrial septal defect may resemble the Eisenmenger complex, although usually there is greater right ventricular enlargement and evidence of pulmonary plethora with prominent peripheral vessels. The absence of cyanosis and arterial oxygen unsaturation are important differential points. When marked pulmonary hypertension develops in the presence of an atrial septal defect, the cardiac contours may be very similar to those of the Eisenmenger complex. Rarely, the hypertension may be so marked as to cause a right-to-left shunt.

b. Ventricular septal defect.[26,35] The classical maladie de Roger is unlikely to be confused with the Eisenmenger complex, since the heart is normal in size and configuration. A large ventricular septal defect, however, offers more of a roentgenologic problem. There is usually bi-ventricular enlargement with more obvious left ventricular prominence than in the Eisenmenger complex. Engorgement of the pulmonary vessels is marked and extends to the periphery. Evidence of right atrial enlargement is rare. The absence of cyanosis or of significant arterial oxygen unsaturation is important. If the pulmonary artery and the right ventricular pressure increase markedly, a right-to-left shunt may develop and then this anomaly is identical with the Eisenmenger complex.

c. Patent ductus arteriosus. The common variety of patent ductus arteriosus offers little difficulty in differential diagnosis on clinical grounds alone. In the presence of pulmonary hypertension,[21] however, the distinction may be more difficult because enlargement of the right ventricle and greater dilatation of the pulmonary artery then occur, and the typical murmur may be replaced by a systolic murmur alone. Under these circumstances, angiocardiography, cardiac catheterization, and differential blood oxygen determinations from arteries of the upper and lower extremities may be required.

d. Primary pulmonary hypertension.[16,25] The roentgen appearance of the heart and lungs in primary pulmonary hypertension is similar to that in the Eisenmenger complex. Indeed, the disparity between central and peripheral vessels may be more marked in idiopathic pulmonary hypertension. Cyanosis is rare, except pre-terminally, dyspnea marked. Angiocardiography and peripheral blood oxygen studies are important for differentiation.

Such other conditions as the Bing Taussig syndrome, a trilocular heart, chronic pulmonary thrombosis, cor pulmonale, and even (rarely) mitral stenosis must also be excluded at times. When cyanosis is profound and peripheral vascularity significantly diminished in childhood, the tetralogy of Fallot with valvular stenosis may be considered. Careful evaluation of the central pulmonary arteries demonstrates that they are normal or small in size in this anomaly, in contrast to the Eisenmenger complex.

SUMMARY AND CONCLUSIONS

1. The clinical, physiologic, and roentgenologic features of the Eisenmenger complex have been evaluated.

2. In a series of 12 cases, the most con-

stant roentgenologic features were right ventricular enlargement, enlargement of the main and hilar pulmonary arteries, and disparity between the large size of the proximal pulmonary arteries and the small caliber of the peripheral pulmonary arteries. The aorta appeared hypoplastic in most instances.

3. The roentgenographic appearance associated with pulmonary hypertension in congenital heart disease is frequently distinctive; this is well illustrated by studies of the Eisenmenger complex.

4. Differential diagnostic criteria and the role of angiocardiography have been briefly considered.

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ROENTGEN MANIFESTATIONS OF UNPERFORATED AORTIC SINUS ANEURYSMS*

REPORT OF THREE NEW CASES

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THE aortic sinuses are three small dilatations at the root of the aorta. Because of their intracardiac location, they are invisible during conventional roentgenography. With the advent of contrast visualization of the aorta, recognition of the aortic sinuses, especially aneurysms, became possible. So far, 20 cases of unperforated aortic sinus aneurysms have been recognized during life at this center. Seventeen have recently been reported;[5,12,16,18,19] the eighteenth, nineteenth and twentieth cases are herein described and the roentgen features which established the diagnosis are reviewed.

Aneurysms of the aortic sinuses (of Valsalva) are rare and are either congenital or acquired. The acquired types are mostly due to syphilis or bacterial endocarditis. The congenital are believed to be due to a developmental defect in either the aorticopulmonary septum or the elastic tissue of the aortic sinuses.

Jones and Langley,[9] in a classic paper in 1949, reviewed the literature; they recorded 43 autopsy proved cases and added 4 new ones. Warthen in 1949 reported the rupture of a congenital right aortic sinus into the left ventricle.[21] Venning[20] in 1951 reported 3 additional cases. Burchell and Edwards,[3] also in 1951, described a congenital aortic sinus which perforated into the right heart; a ventricular septal defect was also present. Falholt and Thompson[7] in 1953 in describing a case of unruptured congenital right aortic sinus aneurysm diagnosed by retrograde aortography increased the number of congenital sinus of Valsalva aneurysms to 34. In 1954, Besabe, *et al.*[1] added a case of congenital aortic sinus aneurysm with rupture into the right atrium due to bacterial endocarditis which also caused embolic gangrene of a foot. Oram and East[13] recently reported sudden rupture of congenital aortic sinus aneurysms in 2 men; the diagnosis was confirmed by cardiac catheterization in one. An attempt to repair the rent failed. Brown, *et al.*[2] also operated on a patient who had rupture of an aneurysm and endocarditis which were diagnosed clinically and by cardiac catheterization. The patient lived fourteen days. Nineteen cases of syphilitic aneurysm of the aortic sinuses have been reported in the English literature since 1914; the antemortem diagnosis was made only once.[12]

REPORT OF CASES

CASE 1. *Congenital Aortic Sinus Aneurysm with Aortic and Mitral Stenosis.* A seven year old male (New York Hospital No. 721959) was referred by Dr. Hyman Bass on June 13, 1955, because of exertional dyspnea and easy fatiguability of two years' duration. The child was well until the age of three years when a heart murmur was heard following pertussis. Fever, joint or muscle pains, tonsillitis and chorea were denied.

On examination the patient was well developed and well nourished: weight, 60 pounds; height, 48½ inches. Only the cardiovascular system was abnormal. The heart was enlarged to percussion; the rate was 100 and regular and the blood pressure was 130/60 mm. Hg. Systolic thrills were felt over the apex of the heart and aortic area and loud systolic murmurs were heard over these areas. A presystolic murmur was also heard over the apex of the heart. The first heart sound was louder than the second; the second pulmonic sound was louder than the aortic. The femoral arterial pulses were palpable.

Laboratory data were as follows: hemoglobin, 12 gm./100 cc.; erythrocytes, 5.1 m/cu. mm.;

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the white blood cell count and differential smear were normal. The electrocardiogram showed no deviation of the electrical axis; the heart was in the intermediate position; the limb, precordial and vector leads were abnormal suggesting right auricular and left ventricular enlargement.

Roentgenographic study (Fig. 1*A*) revealed enlargement of the heart, especially the left atrium and ventricle. Angiocardiography (Fig. 1*B*) showed some enlargement of the right atrium, ventricle, pulmonary artery and branches. The left atrium was enlarged (Fig. 1*C* and 1*D*) and measured 7 by 6 cm. The left ventricle chamber and wall were also markedly enlarged and there was aneurysmal dilatation of the aortic sinuses which measured 27 mm. in diameter. The ascending aorta appeared mildly dilated.

CASE II. *Syphilitic Saccular Aneurysm of the Ascending Aorta with Diffuse Aneurysmal Involvement of the Aortic Sinuses, Aortic Regurgitation and Congestive Heart Failure.* A forty-five

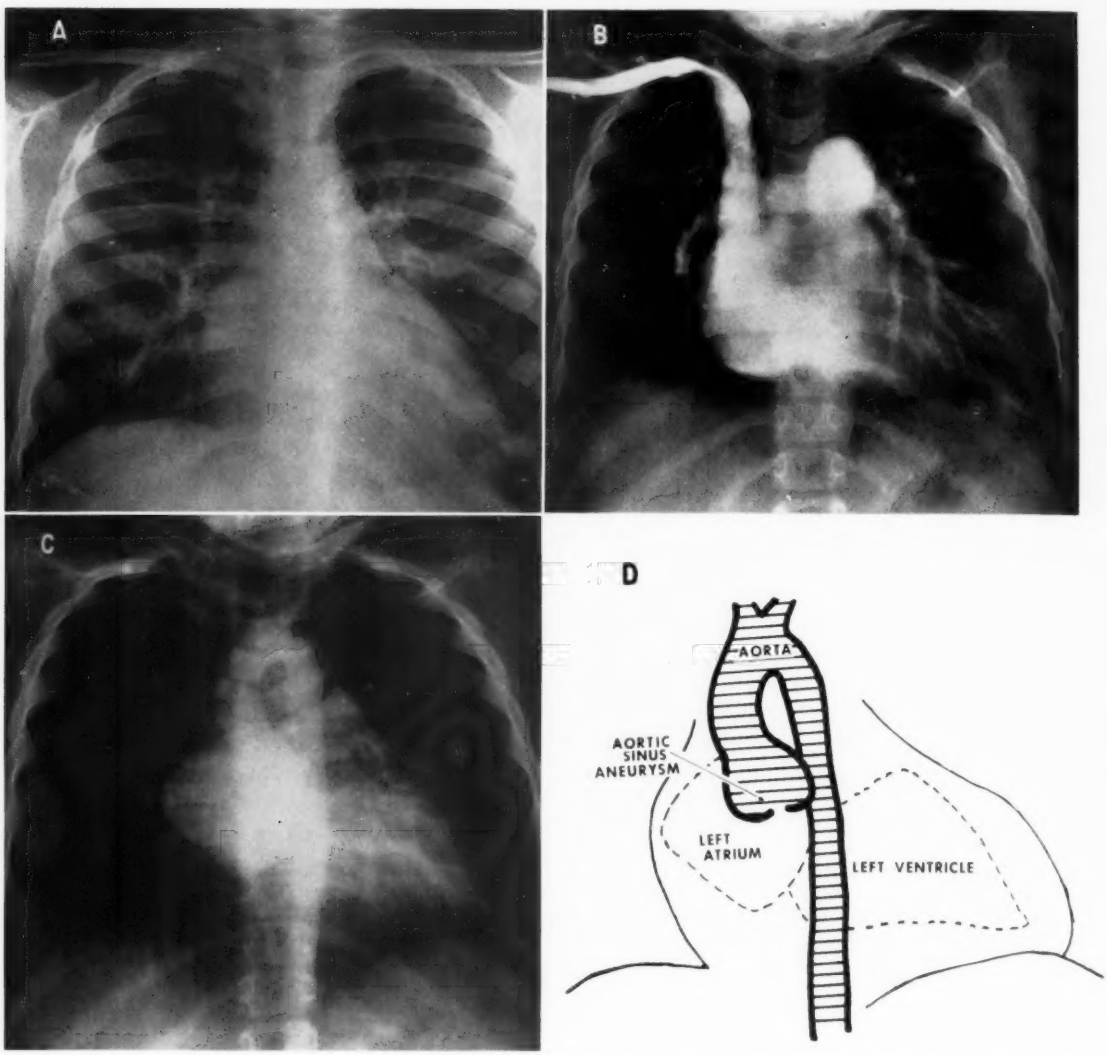

FIG. 1. Case 1. Congenital aortic sinus aneurysm associated with aortic and mitral stenosis in a seven year old boy. (*A*) Conventional frontal roentgenogram shows enlargement of the heart, especially the left ventricle. (*B*) Frontal angiocardiogram at 1½ seconds discloses some enlargement of the right atrium. (*C*) Frontal angiocardiogram at 5½ seconds reveals an enlarged left ventricle (chamber and wall) and left atrium. There is also aneurysmal dilatation of the aortic sinuses. (*D*) Tracing of *C*.

year old Negro (New York Hospital No. 713268) was referred for angiocardiography. The patient had attended the Roosevelt Hospital since 1951. At that time he was found to have congestive heart failure due to aortic insufficiency. After treatment with 17.5 million units penicillin, bed rest and digitalis, he improved. In February, 1955, he was re-admitted because of dyspnea, orthopnea, ankle edema, and bouts of paroxysmal nocturnal dyspnea. Fluoroscopy disclosed aneurysmal dilatation of the ascending aorta, and physical examination showed the heart to be enlarged with regular sinus rhythm, rate, 86. The blood pressure in the left arm was 150/30; in the right arm it was 90/30. There were grade 4 systolic and diastolic murmurs at the apex and base of the heart. A diastolic shock was also felt over the precordium. The liver was tender and enlarged and a Corrigan pulse was present.

Electrocardiographic examination showed occasional ventricular premature contractions and left ventricular hypertrophy. Serological tests of the blood for syphilis were negative. Roentgenographic examination of the chest showed marked enlargement of the heart and huge aneurysmal dilatation of the ascending aorta (Fig. 2*A*). Angiocardiography was done on June 21, 1955 (Fig. 2, *B*, *C*, *D* and *E*), and revealed aneurysmal dilatation of the ascending aorta involving the aortic sinuses. As a result, there was marked displacement of the terminal end of the superior vena cava and of the inflow tract of the right ventricle (Fig. 2, *B* and *C*).

On July 14, 1955 (at another hospital), an attempt was made to alleviate the aortic regurgitation and congestive heart failure by the installation of a Hufnagel plastic valve. The patient died postoperatively.

Postmortem examination* showed a huge heart: weight, 765 grams. The left ventricle was markedly hypertrophied. There was a large aneurysmal dilatation of the ascending aorta which involved the sinuses of Valsalva. In this area the aorta measured 10 cm. in greatest diameter (Fig. 2*F*). A dissecting aneurysm of recent origin was also present throughout the ascending aorta. The aortic valve measured 8 cm. in circumference; each leaflet showed a sharp thickening along the free margin of the cusps. The ostia of the coronary arteries were, however, widely patent. The intima of the aorta in the region of the aneurysm showed a mottled and wrinkled yellowish gray appearance. The mitral and tricuspid valves were dilated and showed slight thickening at the free margins. There was passive hyperemia of the legs, liver, kidneys and spleen. A recent myocardial infarction was also present.

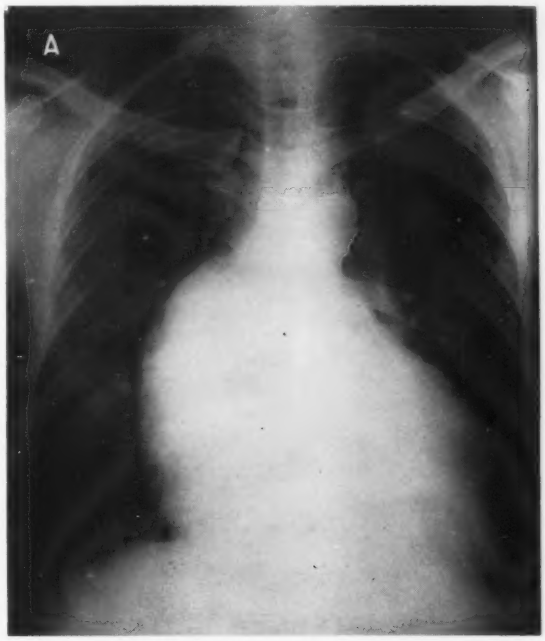

FIG. 2. Case II. Syphilitic saccular aneurysm of the ascending aorta with aneurysmal involvement of the aortic sinuses in a forty-five year old man with aortic regurgitation and congestive heart failure. (*A*) Conventional frontal roentgenogram shows marked enlargement of the heart, especially the left ventricle. There is a huge saccular aneurysm of the ascending aorta which extends into the heart.

CASE III. *Syphilitic Aortic Sinus Aneurysm with Aortitis, Aortic Regurgitation and Congestive Heart Failure.* A sixty-one year old Negro laborer was admitted to the Bronx Veterans Administration Hospital on January 25, 1955, complaining of dyspnea. For over a year he had had increasing dyspnea on exertion associated with cough and edema of the legs. For several months he also had anorexia, pain in the right upper abdomen and had lost thirty pounds in weight. He recalled a penile lesion during his youth. Physical examination revealed sluggish reactions of the pupils to light. Moist rales were present at the bases of the lungs. The heart was enlarged; the position of the maximum impulse of the heart was in

* Courtesy of the Department of Pathology, The Roosevelt Hospital, New York, New York.

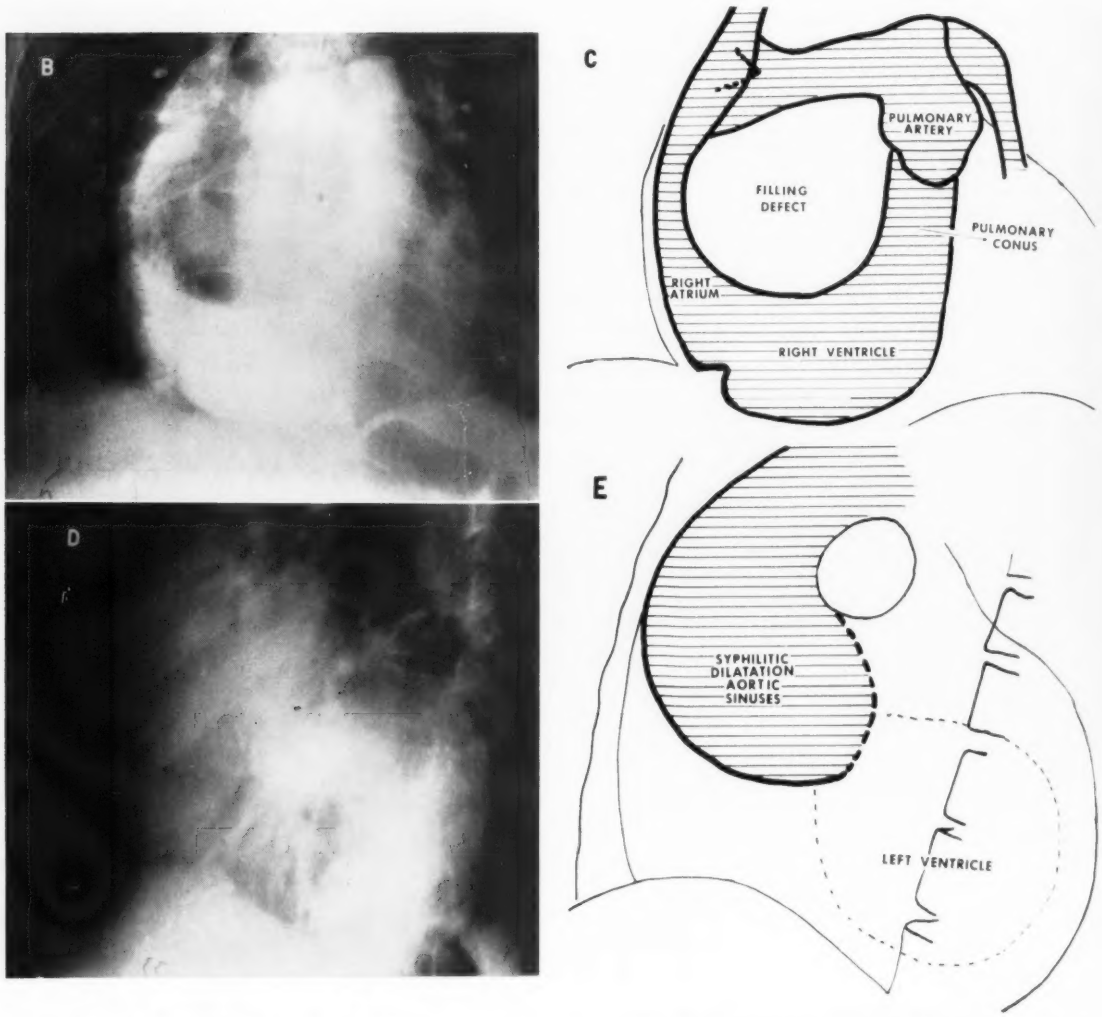

FIG. 2. Case II. (*B*) Frontal angiocardiogram at 5½ seconds shows marked displacement and deformity of the lower end of the superior vena cava, right atrium and ventricle by a huge intracardiac structure. (*C*) Tracing of *B*. (*D*) Left anterior oblique angiocardiogram at 33 seconds shows the tremendous saccular aneurysm of the aorta extending downward and including the aortic sinuses. (*E*) Tracing of *D*.

the sixth intercostal space in the anterior axillary line. There was a harsh (grade 3) systolic murmur at the apex transmitted to the axilla and a blowing diastolic (grade 3) murmur at the base of the heart, loudest at Erb's point. The second aortic sound was louder than the second pulmonic sound. The rate was 100, regular with occasional premature contractions. The blood pressure was 120/52. The liver was tender and enlarged four finger breadths below the costal edge and there was a three plus edema of the legs.

Laboratory data revealed a positive serologic test of the blood for syphilis. The electrocardiogram showed right bundle branch block and left ventricular hypertrophy. Roentgen examination of the chest revealed the heart to be markedly enlarged, especially the left ventricle (Fig. 3). Calcification of the ascending aorta was evident and the calcifications were seen to extend into the intracardiac origin of the aorta (Fig. 3*A*). Aneurysmal dilatations of the aortic sinuses could be recognized by the deposits of calcium in the walls of the aortic sinuses, especially in lateral view (Fig. 3*B*).

The patient remained in the hospital for five months and improved with rest, digitalis and penicillin therapy. He was re-admitted on September 12, 1955, because of the recurrence of severe dyspnea, right upper quadrant pain and

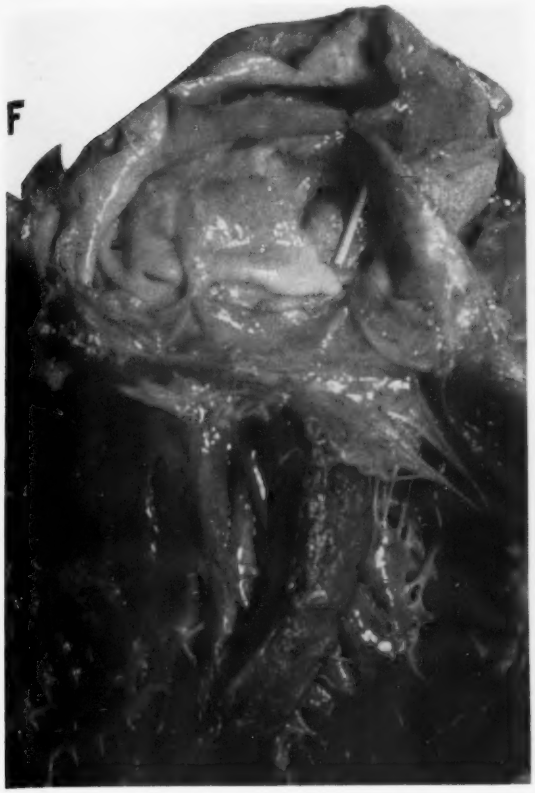

Fig. 2. Case II. (*F*) Postmortem specimen showing the aortic aneurysm involving the aortic sinuses. Note the hypertrophy of the left ventricle and trabeculae carnae.

leg edema. This time the pulmonary congestion and the liver and leg edema proved refractory to treatment and he died in congestive heart failure on January 8, 1956. Consent for an autopsy could not be obtained.

DISCUSSION

NORMAL ANATOMY OF THE AORTIC SINUSES

The aortic sinuses are three small dilatations in the wall of the aorta immediately above the aortic valves. Each sinus lies just above the attachment of an aortic cusp. The right and left coronary arteries usually originate within two of the aortic sinuses; however, they may emerge immediately distal to the aortic sinuses. The aortic sinuses and their corresponding aortic valves are named according to the source of the coronary arteries. Thus, the right and left coronary arteries emerge from the right and left aortic sinuses respectively. The remaining aortic sinus usually lies posteriorly, does not contain a coronary artery and according to the newest nomenclature is designated the noncoronary sinus.[9,13]

The aortic sinuses are intracardiac and cannot be identified on conventional roentgenography. During angiocardiography they appear as small dilatations at the root of the aorta immediately above the aortic valves (Fig. 4, *A* and *B*) and are best visualized in the left anterior oblique projection.[4] Inconstant filling of the coronary arteries during angiocardiography does not allow complete identification of the sinuses. However, in the left anterior oblique view, the right coronary sinus is anterior and just behind the sternum. The aortic sinuses of Valsalva are in close relation to all the cardiac chambers, particularly the right atrium and ventricle. The superior vena cava, the pulmonary artery, the left ventricle and the left atrium are adjacent.

CLINICAL FEATURES OF UNPERFORATED CONGENITAL AORTIC SINUS ANEURYSMS

Clinically, there is only a single clue to the presence of an unperforated congenital aortic sinus aneurysm. It consists of a harsh systolic murmur (grade 3 to 4) over the heart, especially to the left of the sternum. Such a murmur, if associated with coarctation of the aorta,[5] anomalies of the aortic valves or arch,[16,18] or arachnodactyly[19] should arouse suspicion of an aortic sinus aneurysm. Confirmation can then be had by angiocardiography.

ROENTGENOGRAPHY OF CONGENITAL UNPERFORATED AORTIC SINUS ANEURYSMS

Conventional Roentgenography. On fluoroscopy, cases of congenital aortic sinus aneurysms frequently exhibit unusual pulsation of the root and ascending aorta. However, when there is hypertension, a dilated ascending aorta, due to associated coarctation of the aorta or aortic valve disease, the value of this observation is limited. An abnormal and enlarged cardiac sil-

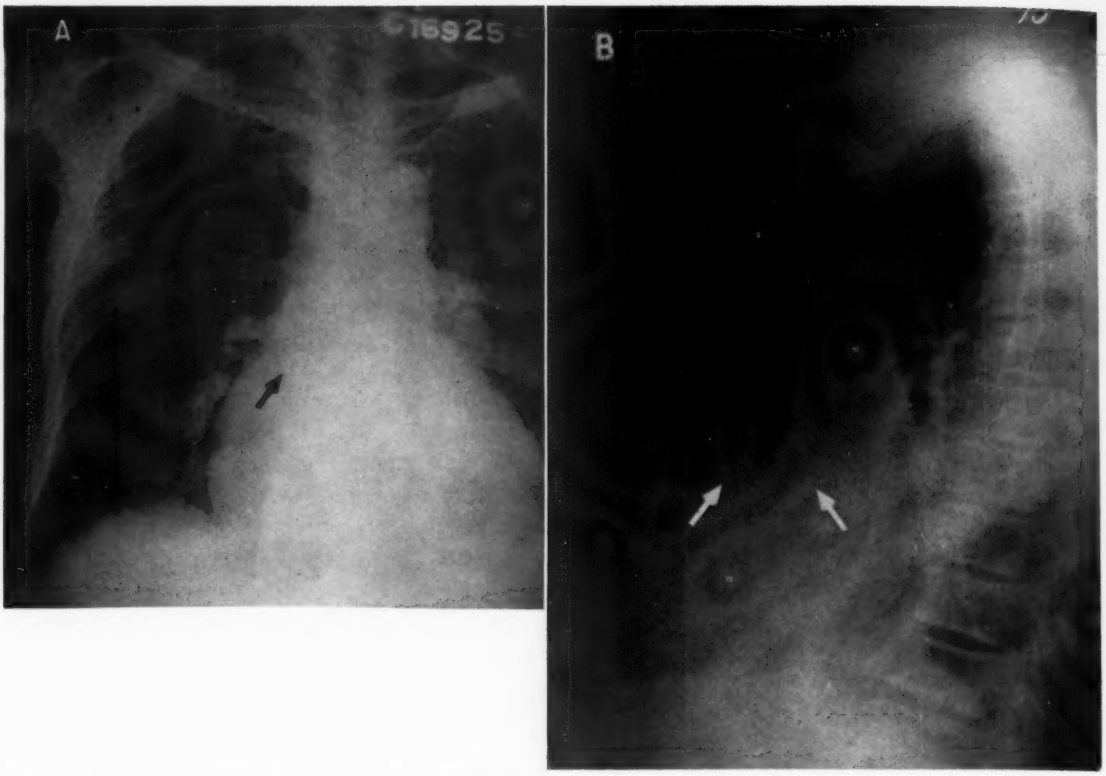

FIG. 3. Case III. Syphilitic aortitis with aortic sinus aneurysm, aortic regurgitation and congestive heart failure in a sixty-one year old man. (*A*) Conventional frontal roentgenogram shows enlargement of the heart, especially the left ventricle. Arrow points to intracardiac calcification continuous with the ascending aortic calcification. (*B*) Lateral roentgenogram also shows calcium (arrows) outling the dilated aortic sinuses.

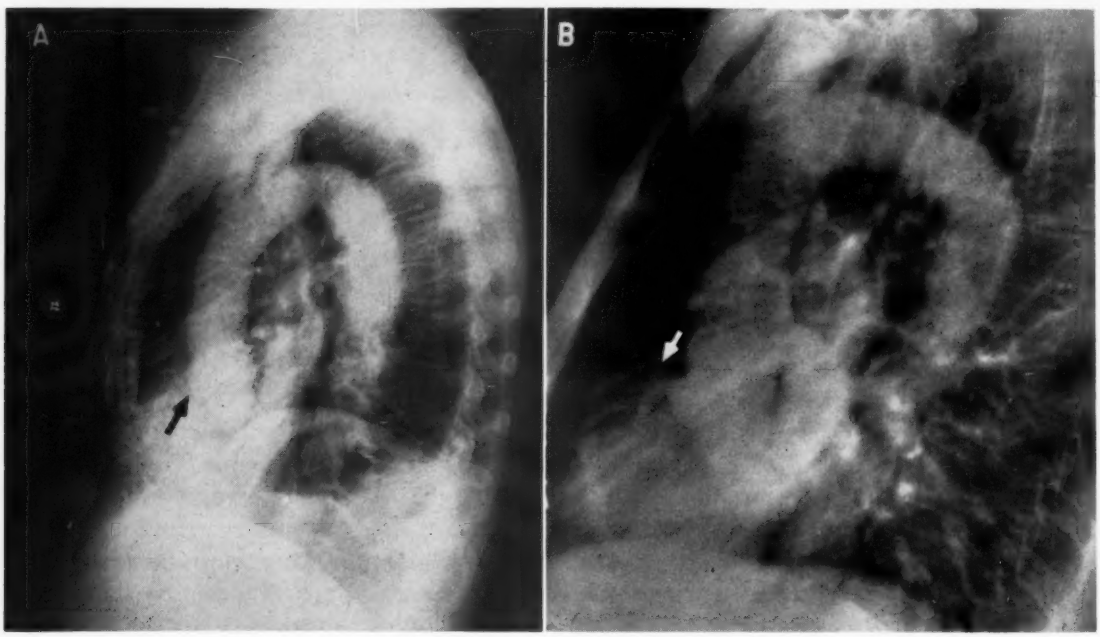

FIG. 4. The Normal aortic sinuses. (*A*) Left anterior oblique angiocardiogram reveals the aortic sinuses. Arrow points to the right aortic sinus. (*B*) Left anterior oblique angiocardiogram shows the right aortic sinus just below the right coronary artery. (arrow)

houette may be expected in the roentgenograms of patients with congenital aortic sinus aneurysms. However, in 2 instances, a patient with pseudocoarctation[16] and a patient with arachnodactyly had normal hearts.[19] In addition, 4 patients with coarctation of the aorta exhibited rib notchings, an enlarged left ventricle, and evidence of poststenotic enlargement of the descending aorta verified by indentation of the esophagus.[5] The patient with pseudocoarctation had a prominent shadow in the region of the aortic knob and also a dilated descending aorta; however, rib notching was not present.[16] Two of 3 patients with arachnodactyly had enlarged left ventricles.[19] In the patient with aortic and mitral valvular stenoses there was enlargement of all cardiac chambers (Fig. 1).

Angiocardiography. Angiocardiography provides the definitive diagnosis of aortic sinus aneurysm. Generalized aneurysmal dilatation of all the aortic sinuses was present in all but 1 instance. In that case, only the right aortic sinus was involved.[18] In coarctation of the aorta, the point of coarctation, the dilated brachiocephalic and collateral arterial branches, as well as the enlarged left ventricle, were clearly outlined.[5] In contrast, in the pseudocoarctation case, although there was an unusually dilated left subclavian artery, an apparent point of coarctation, and a dilated descending aorta, there was no collateral arterial circulation and the heart was not enlarged.[16]

SYPHILITIC UNPERFORATED AORTIC SINUS ANEURYSMS

Pathologic Aspects. Syphilitic involvement of the cardiovascular system is commonest at the aortic valve and ascending aorta. It begins with weakening and destruction of the intimal surface of the aorta and elastic fibers of the media. When aortitis becomes manifest, either a diffuse aneurysmal dilatation or a localized saccular aneurysm of the ascending aorta may occur. Calcium plaques are frequently deposited in the damaged intima; their recognition in the root and ascending aorta becomes an important clue of syphilis.[15,22] If the aneurysm is large, stasis of blood occurs and a laminated thrombus partially fills the lumen. The aortic sinuses may similarly be involved and aneurysmal dilatation or sacculation of the sinuses results. Dilatation of the aortic ring and the production of of aortic insufficiency are usual.[12]

Unlike congenital and mycotic aneurysms of the aortic sinuses which are paper thin and prone to rupture,[9] syphilitic aneurysms usually become very large and laminated before they perforate.[9,15] Any of the aortic sinuses may be the site of origin of a syphilitic aneurysm. This is in contrast to the congenital type which is believed to involve chiefly the right and noncoronary sinuses.[9,13] Contrary to reports, there is no predictable relation between the sinus of origin and the structure into which an aneurysm perforates.[14] Eight of the 11 perforated cases reported in the literature (73 per cent) ruptured into the lesser circulation (pulmonary artery, 4; right atrium, 2; and right ventricle, 2), and produced a left to right shunt.[12] Syphilitic aortic sinus aneurysms, unlike the congenital, may also rupture outside the heart. This is probably because the syphilitic aneurysms attain such a large size.

In the literature as well as in our cases, the most common cause of death in patients with syphilitic aortic sinus aneurysm was heart failure due to aortic insufficiency. Rupture of the aortic sinus aneurysm in 8 of the reported cases with the establishment of aorticocardiac or aorticopulmonary shunts hastened failure; none survived more than two months. On the other hand, rupture outside the heart caused sudden death. In 3 of the reported cases there were coronary artery occlusions due to pressure of the adjacent aortic sinus aneurysms. Myocardial infarction was found distal to the occluded left coronary artery and caused sudden death in one instance, while in another case reported in the literature, the aneurysm was intact but bulged into the interventricular septum and pulmonary conus causing pulmonary stenosis and delayed atrioventricular conduction, presumably due to pressure on the bundle of His.[12]

ROENTGENOGRAPHY OF SYPHILITIC UNPERFORATED AORTIC SINUS ANEURYSMS

Conventional Roentgenography. The diagnosis of syphilitic aortic sinus aneurysm can often be made after conventional roentgenography. A large boot shaped heart with a large left ventricle caused by the isolated aortic insufficiency was present in all but one case. Pulsatile dilatation of the ascending aorta and fusiform or saccular aneurysms of the ascending aorta are also frequent findings. Linear calcification of the aorta is common and has long been recognized as an important sign of syphilitic aortitis.[15,22] When the calcification extends into the intracardiac origin of the aorta and outlines the aortic sinuses (Fig. 3,*A* and *B*) or becomes massive in this region, the diagnosis of syphilitic aortic sinus aneurysm becomes almost a certainty.

Angiocardiography. Aortic sinus aneurysms are readily demonstrated by angiocardiography. In the syphilitic, two types are recognized. In 8 instances, the aneurysms were continuations of huge fusiform or saccular aneurysms of the ascending aorta (Fig. 2); in 4, the aneurysms were localized at the aortic sinuses and associated with aortitis (Fig. 3).[12] In all instances the syphilitic aneurysms were considerably larger than the congenital types and, therefore, created pressure effects in the surrounding cardiac chambers and great vessels (Fig. 2,*B* and *C*). There are 2 previously reported cases, one after necropsy,[15] the other still alive[12] in which deformity of the pulmonary conus, pulmonary artery and its left branch was sufficient to cause right ventricular hypertrophy and chronic cor pulmonale.

Although angiocardiography was not performed in Case III and autopsy confirmation is lacking, the characteristic location of the calcium deposits in the ascending aorta extending into the aortic root and outlining the aortic sinus with calcium warrants the diagnosis of aortic sinus aneurysm associated with syphilitic aortitis (Fig. 3). Such was the case, although complicated by a saccular aneurysm of the ascending aorta, in a patient recently reported (Fig. 5).[12]

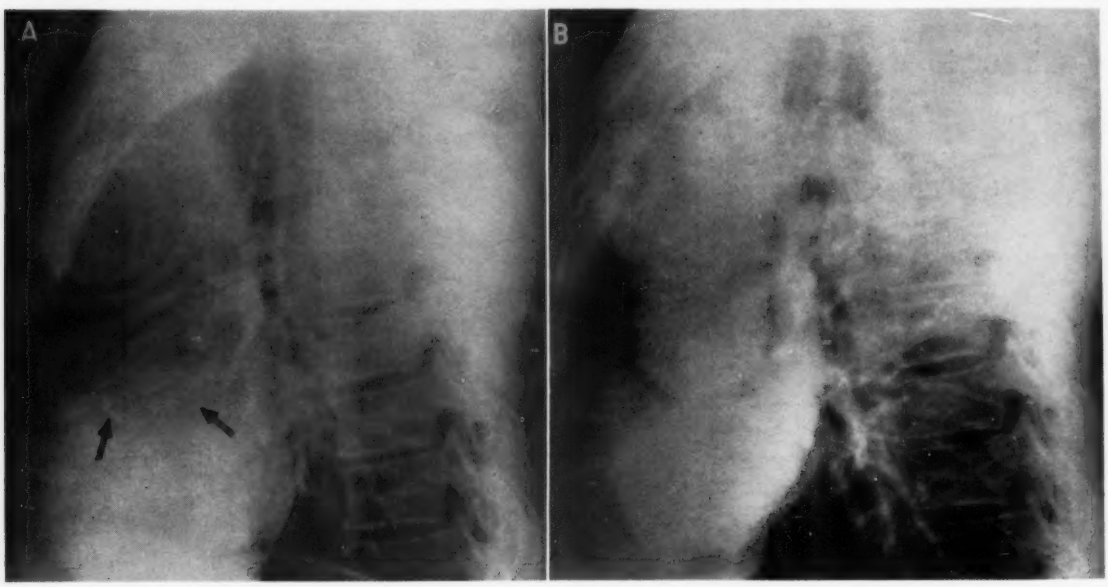

FIG. 5. Syphilitic aortic sinus aneurysm associated with a saccular aneurysm of the ascending aorta. (*A*) Conventional roentgenogram reveals a saccular ascending aortic aneurysm which extends to the aortic root and involves the aortic sinuses (outlined by calcium deposits [arrows]). (*B*) Lateral angiocardiogram demonstrates the aneurysmal dilatation of the aortic sinuses and the huge ascending aortic aneurysm. (Previously published.[12])

Figure 5*A* shows the calcium deposit in the ascending aorta extending into the root of the aorta and clearly outlining the aortic sinuses. On a lateral angiocardiogram, the syphilitic aortic sinus aneurysm associated with the aneurysm of the ascending aorta is readily seen (Fig. 5*B*).

RUPTURE OF AORTIC SINUS ANEURYSMS

Sudden onset of severe dyspnea, right heart overloading and failure, enlargement of the pulmonary artery and evolution of machinery-like murmurs may mean rupture of an aortic root aneurysm into the pulmonary artery or right heart chambers.[2,8,13] Confirmation may be had by cardiac catheterization.[2,6,11,13] Repairs of ruptured aortic sinus aneurysms were attempted in 3 instances but were unsuccessful.[2,10,13]

TREATMENT AND PROGNOSIS OF AORTIC SINUS ANEURYSMS

Surgical repair of syphilitic sinus of Valsalva aneurysms is a formidable undertaking because of their large size and serious pressure effects on the neighboring cardiovascular structures. However, in the rare congenital type when the aneurysm is localized to a single aortic sinus, approach through the right atrium or ventricle appears feasible.[2,13] In one of our recent cases, surgical repair of the aneurysm appeared especially dangerous because of the associated aortic regurgitation, coarctation of the aorta and the recent bacterial endocarditis. However, the patient withstood surgical correction of the aortic coarctation very well.[18] Even though congenital sinus of Valsalva aneurysms are paper thin and tend to rupture,[9] 9 cases remained unperforated during a three to five year period of observation.[17] At the present time surgical repair of the diffuse types of unruptured congenital sinus of Valsalva aneurysms does not seem warranted.

Early diagnosis and treatment of syphilis with penicillin will prevent aortitis and aneurysms. Of our 11 cases of syphilitic aortic sinus aneurysm, 4 are dead, 3 are lost to observation and 4 are alive and well and have been under medical care for periods up to five years.

The 9 patients with congenital types of aortic sinus aneurysms are all alive. The 4 with coarctation of the aorta had resection of the coarcted segments with complete relief of the hypertension after end to end anastomosis of the aorta; all the patients are well enough to work. One of the 3 patients with arachnodactyly, however, is still troubled by mild congestive heart failure; the other 2 continue to be free of cardiovascular complaints.[19] The patient with pseudocoarctation is also free of cardiovascular symptoms and is working.[16] The child with the multiple valve disease and heart enlargement remains handicapped.

The long term prognosis of patients with unperforated aortic sinus aneurysms is unknown. Even though there have been only a few years of observation, the general well being of the majority of the patients indicated that the prognosis is not immediately fatal. Once the aneurysm ruptures, the situation is usually hopeless. Therefore, even though the surgical approach is difficult and hazardous and despite the desperate circulatory hemodynamics, an attempt to repair the perforation should be made.[17]

SUMMARY AND CONCLUSION

Three new cases of unperforated aortic sinus aneurysms were diagnosed antemortem by roentgenographic studies. In the first, a child of seven years, the aortic sinus aneurysm is believed to be of congenital origin and was associated with mitral and aortic stenosis. The aortic sinus aneurysm was unsuspected clinically. The 2 other cases had acquired syphilitic aortic sinus aneurysms. Both patients had chronic congestive heart failure due to aortic regurgitation. One also had a huge aneurysmal dilatation of the ascending aorta which involved the aortic sinuses. Diagnosis was established by angiocardiography and confirmed by postmortem examination

of the heart and aorta. In the other, the appearance of calcification in the walls of the dilated ascending aorta with extension of the calcium deposits into the root of the aorta outlining the aortic sinuses was considered characteristic enough to establish the diagnosis of syphilitic aortitis and aortic sinus aneurysm.

Angiocardiography provides the definitive method of establishing the diagnosis of unperforated aortic sinus aneurysms. However, the majority of the syphilitic type has characteristic conventional roentgenographic findings. These consist of linear calcification of the walls of the ascending aorta (a reliable sign of syphilitic aortitis) extending into the root of the aorta and often outlining the aortic sinuses. In some instances massive calcifications at the aortic root represent calcified thrombosis of an aortic sinus aneurysm.

When perforation of an aortic sinus aneurysm into the right cardiac chambers occurs, there are usually classical symptoms of sudden onset of chest pain and dyspnea, soon followed by relentless heart failure. On physical examination a high pitched continuous musical murmur can be recognized. Often there are roentgen signs of superior vena caval, right atrial and right ventricular dilatation frequently followed by right ventricular enlargement. Confirmation of an aorticocardiac shunt can be secured by right heart catheterization which will disclose highly oxygenated blood and hypertension in the right cardiac chambers and pulmonary artery.

Repair of a ruptured congenital aortic sinus aneurysm should be attempted especially if there is a chronic and latent phase of the aorticocardiac shunt. Repair of a ruptured syphilitic aortic sinus aneurysm seems unwarranted for, in addition to the new aorticocardiac shunt, there is the burden of long standing disease with hypertrophy of the left ventricle because of aortitis and aortic regurgitation.

Roentgenography is essential for establishing the diagnosis of aortic sinus aneurysms and permits their classification into the congenital and acquired (syphilitic) types. It also provides important information when rupture occurs and is useful for the selection of the case that may be amenable to surgical repair of the aorticocardiac shunt.

ADDENDUM

Since this paper was submitted for publication, Case 1 was re-admitted to the hospital because of bacterial endocarditis. Recovery followed antibiotic treatment.

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ROENTGENOGRAPHIC VISUALIZATION OF THE CORONARY ARTERIES

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IT IS well known from autopsy statistics that calcification of the coronary vessels is very common, notably in the older age group. However, in most instances the deposits are not identifiable on roentgen films. Presumably, the arteriosclerotic process must be advanced and rather dense to become visualized. Other considerations which play a definite role in the roentgenographic recognition of coronary sclerosis are (1) awareness of the common sites and characteristics of such densities, (2) good technical quality of roentgenograms with multiple projections including posteroanterior, both oblique and lateral views, (3) differentiation from other intra- and extra-cardiac densities, such as calcification of the pericardium, valves, annulus fibrosus, costal cartilages, hilar lymph nodes, and pulmonary vessels projected through the cardiac borders.

The most common site of visible calcification is in the circumflex branch of the left coronary artery, just beneath the left heart border, in the region of the upper left ventricular contour. In the left anterior oblique view, the circumflex branch may be identified as a circular calcific deposit posteriorly in the auriculoventricular groove, since it is seen in cross-section in this projection. Next in frequency is the descending branch of the left coronary artery in the upper portion of the interventricular groove. Not uncommonly, both of these branches may be calcified. Less frequently, calcification can be seen in the right coronary artery. This is located close to the anterior or right ventricular border in the left anterior oblique projection and somewhat deeper to the posterior heart border in the right anterior oblique projection (Fig. 1).

Characteristically, these calcifications have the appearance of linear streaks and interrupted plaques arranged in parallel segments. Fluoroscopically, the calcified vessels move synchronously with, and in the same amplitude as, the adjacent heart border. The movements are considerably less dramatic than those exhibited by calcified valves and annulus fibrosus.

Six cases (Table 1) associated with definitive roentgen evidence of sclerotic changes of branches of the left coronary artery were observed. Cardiac consultation, 12-lead electrocardiography, teleroentgenography, urinalysis, serologic tests, and blood cell counts were performed in each instance. When necessary, roentgenograms in other views and neurologic consultation were also requested.

REPORT OF CASES

CASE 1. B.W.K., white male sixty-one years old, was treated for duodenal ulcer, with marked pyloric obstruction for eight years. Two months ago, the patient suffered pressing precordial pains and exertional dyspnea. He also had cramping pains in both legs. He worked as a longshoreman until recently. Examination revealed a well developed male of 176 pounds and 70 inches. Both fundi showed retinal angiosclerosis. There was no cardiac enlargement or murmurs. The tones were of good quality. Regular sinus rhythm was present with a rate of 82 per minute. Blood pressure was 138/86. Sclerotic peripheral arteries were palpated.

Laboratory studies: Roentgenograms of the chest revealed no cardiac enlargement, but there was marked calcification of the left

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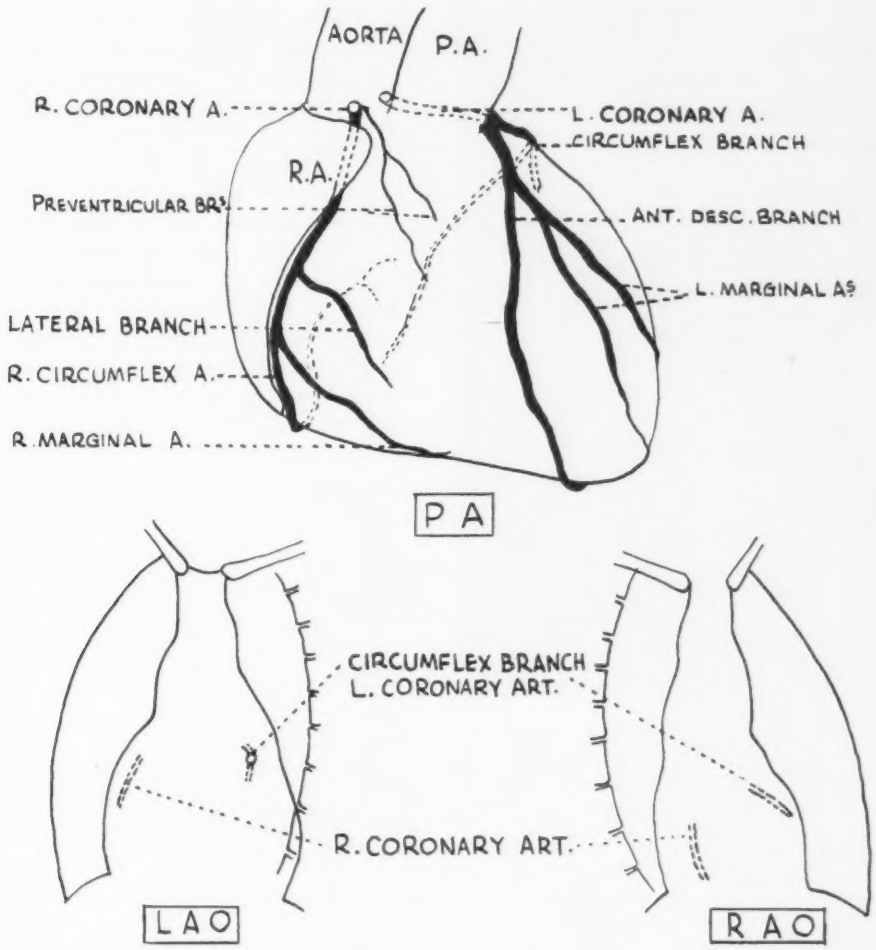

FIG. 1. Distribution of coronary arterial supply and roentgen anatomy in the oblique views.

TABLE I

SUMMARY OF FINDINGS

Case	Age	Sex	Calcifications Visible on Roentgenograms	Electrocardiogram	Symptoms	Blood Pressure
I	61	M	left coronary, circumflex and anterior descending branches	posterior wall infarction	anginal syndrome, dyspnea	138/86
II	64	M	left coronary, anterior descending branch, aorta, leg arteries	negative	headaches, chest pain, dyspnea	162/104
III	56	M	left coronary, circumflex branch, aorta	negative	hemiplegia only	180/120
IV	66	M	left coronary, aorta	negative	tremors, headaches	170/110
V	76	M	left coronary, circumflex and anterior descending branches, aorta, aortic valve	negative	none	130/70
VI	65	F	left coronary, circumflex branch	negative	dyspnea, hemiplegia	210/122

FIG. 2. Case 1. B. W. K. Posteroanterior roentgenogram. Calcification of the left coronary artery and of portions of the circumflex and anterior descending branches.

coronary artery and of portions of the circumflex and anterior descending branches. This was especially visible in the posteroanterior projection (Fig. 2). There was marked calcification of the arch and descending portions of the aorta. Extensive calcification was noted in the abdominal aorta, pelvic and femoral arteries, as well as in the arteries of both lower extremities. The electrocardiographic findings were compatible with an old myocardial infarction of the posterior wall (Fig. 3). Urinalysis, blood cell count and serologic test were normal.

Diagnosis: Generalized arteriosclerosis (predominantly peripheral and cardiac), arteriosclerotic heart disease, old posterior wall myocardial infarction, regular sinus rhythm, anginal syndrome and chronic duodenal ulcer.

Comment. A clinicoroentgenologic correlation is well defined in this patient. Abundant clinical and roentgen evidence of marked generalized atheromatous degeneration was present. Sclerosis of the coronary arteries led to occlusion with infarction of the posterior wall, as indicated on the electrocardiogram. The entire aorta and all branches to the lower extremities were also markedly involved, with symptoms of claudication.

CASE 11. W.A.D., a white male sixty-four years old, suffered from hypertension, exertional dyspnea, and precordial pains for one year. He had been hospitalized for several days because of headaches and dyspnea. He had a herniotomy thirty years previously. He worked as a painter until four years ago. Examination revealed a moderately advanced angiosclerosis of the fundi. There were no cardiac enlargement, murmurs, thrills or rales. The tones were of fair quality, but the second aortic sound was accentuated. There was normal sinus rhythm

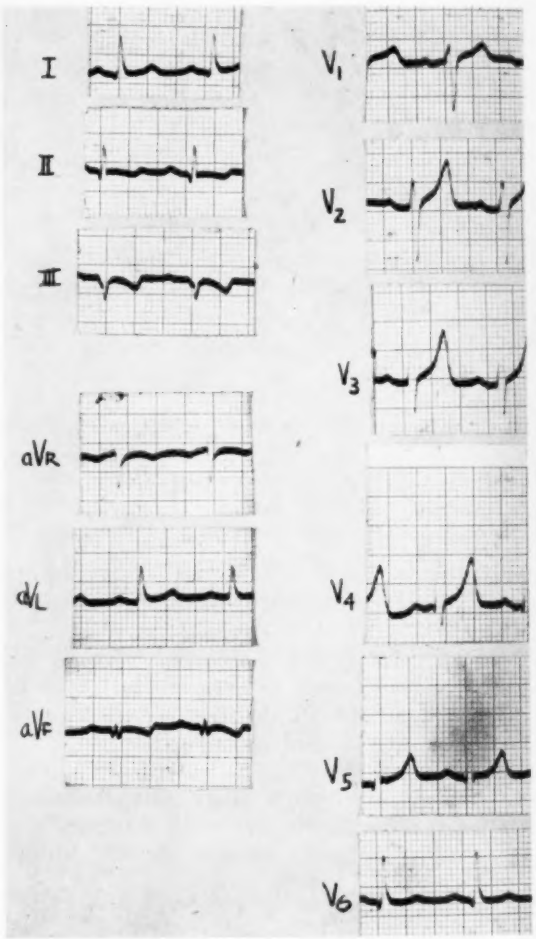

FIG. 3. Changes in leads 2, 3, and aVf typical of old posterior myocardial infarction.

with a rate of 82 per minute. There was no evidence of myocardial insufficiency. The peripheral vessels were sclerotic. Blood pressure was 162/104.

Laboratory studies: Roentgenograms of the chest revealed no cardiac chamber enlargements. Linear calcific streaks were noted at the upper left ventricular contour in the posteroanterior view, following the course of the anterior descending branch of the left coronary artery (Fig. 4). Sclerosis of the aorta and leg vessels was noted. The electrocardiogram, blood cell count and serologic test were normal.

Diagnosis: Generalized arteriosclerosis and mild hypertensive vascular disease. This is another instance of advanced atheromatous degeneration of the cardiovascular system with normal electrocardiogram.

Case III. G.C.F., a white male fifty-six years old, had hypertension for ten years. For this reason he was retired from the Fire Department. He recently suffered two strokes affecting both sides of the body. He improved sufficiently to be able to walk with slight residual incapacity of the right extremities. At present he complains only of occasional headaches. Examination revealed a male of 175 pounds and 67½ inches. There was a Grade 1 hypertensive retinopathy. Cardiac study revealed no enlargement, thrills or rubs. There was a nonlocalized faint systolic murmur heard over the precordium. The second pulmonic sound was greater than the second aortic sound and both sounds were accentuated. The tones were of fair quality. There was no evidence of decompensation. Blood pressure was 180/120. There was regular sinus rhythm, with a rate of 78 per minute. There were mild residuals of right hemiplegia.

Fig. 4. Case II. W. A. D. Posteroanterior roentgenogram. Calcification of the anterior descending branch of the left coronary artery. Calcification is also seen in the aortic arch.

Laboratory studies: Roentgenograms of the chest revealed no evidence of cardiac chamber enlargement. There was tortuosity of the aorta. Heavy calcification of the walls of the circumflex branch of the left coronary artery was visible in the posteroanterior, left anterior oblique and right anterior oblique projections (Fig. 5). In the left anterior oblique projection, the vessel was seen on end in the auriculoventricular groove. The electrocardiogram was entirely normal. Blood cell count, urinalysis and serologic test were normal.

Diagnosis: Old cerebral thrombosis and hypertensive vascular disease.

Comment. Although the electrocardiogram was negative and there was no cardiac symptomatology, fairly marked calcification was evident in the left coronary artery. The cerebral vessels were affected by a similar atheromatous process which culminated in cerebral thrombosis and right hemiplegia. Apparently, the lumina of the coronary arteries were sufficiently patent to obviate clinical or electrocardiographic evidence of myocardial damage or strain pattern.

Case IV. C.I.D., a sixty-six year-old retired white marine engineer, complained of generalized tremors of six months' duration. He also had nervousness, dizziness and headaches. Hypertension was accidentally discovered at this examination. He denied cardiovascular symptomatology other than exertional dyspnea. Examination revealed Grade 2 hyper-

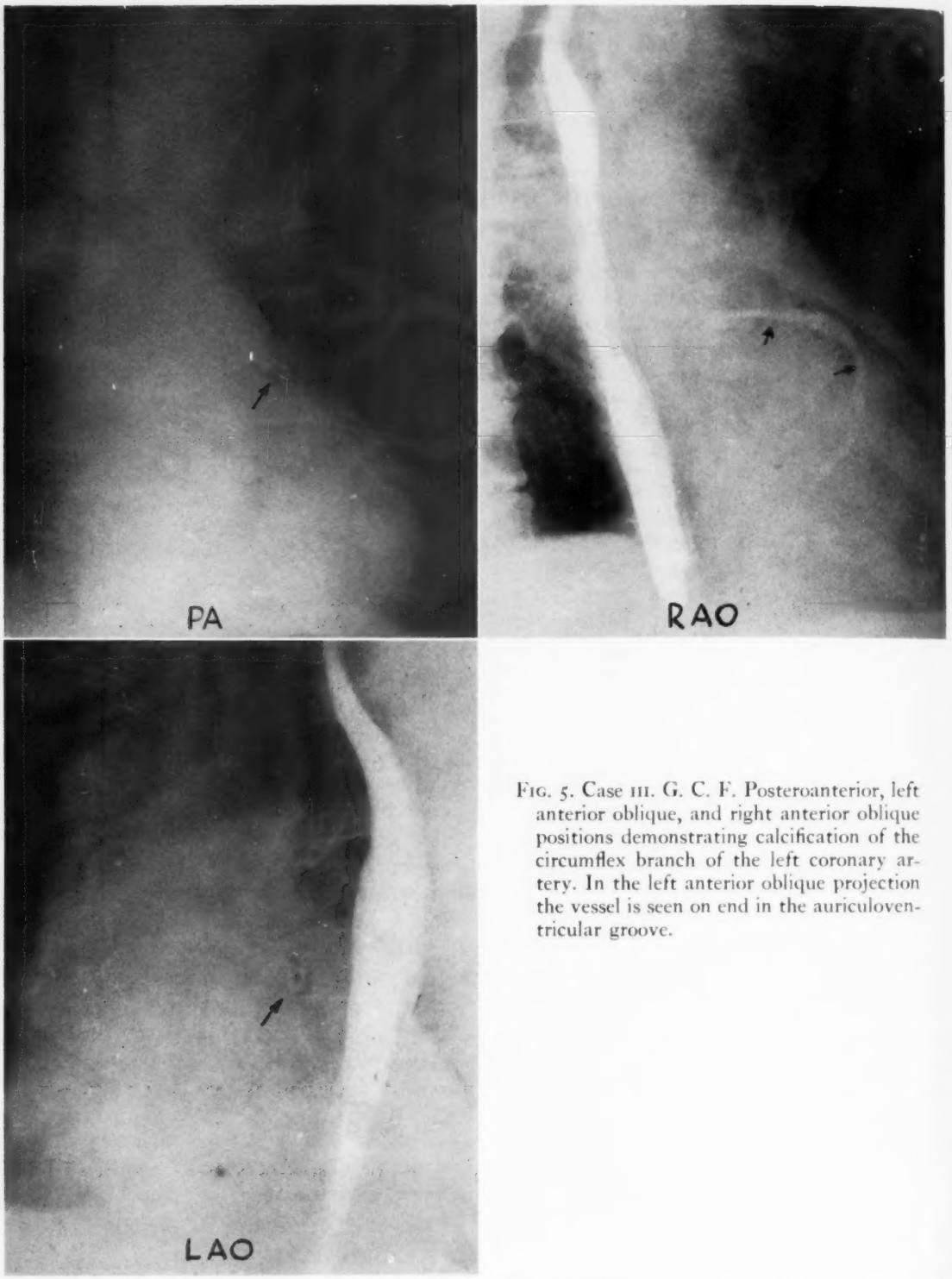

FIG. 5. Case III. G. C. F. Posteroanterior, left anterior oblique, and right anterior oblique positions demonstrating calcification of the circumflex branch of the left coronary artery. In the left anterior oblique projection the vessel is seen on end in the auriculoventricular groove.

tensive retinopathy. There were no cardiac enlargement, thrills, rubs or murmurs. There was normal sinus rhythm with a rate of 80 per minute. There was no evidence of myocardial insufficiency. Blood pressure was 170/110. Neurologic study disclosed characteristic findings of paralysis agitans.

Laboratory studies: Roentgenograms of the chest revealed no cardiac chamber enlargement, but calcific streaks were noted inside the

upper left ventricular border. There was calcification of the arch and descending portions of the aorta. The electrocardiogram was normal, except for occasional premature ventricular contractions. Blood studies, urinalysis and serologic test were normal.

Diagnosis: Hypertensive vascular disease, generalized arteriosclerosis, and paralysis agitans.

Comment. Generalized arteriosclerosis was manifested clinically in the cerebral arteries only. Although there was abundant roentgen evidence of cardiovascular sclerotic changes, there were very few cardiac complaints, and the electrocardiogram was normal.

Case v. McA., a Negro male 76 years old, complained of a productive cough and dyspnea of two weeks' duration. Ophthalmoscopic examination revealed moderate angiosclerosis of the fundi and a post-traumatic adherent leukoma. There were occasional basal rales and rhonchi in both lungs. The heart was not enlarged, and there were no murmurs, but the tones were of poor quality. There was normal sinus rhythm with a rate of 86 per minute. Blood pressure was 130/70. There was no evidence of myocardial insufficiency. The peripheral vessels were thickened and sclerotic.

Laboratory studies: A roentgenogram of the chest revealed no enlargement of the heart. There was extensive calcification of the aortic arch. At the upper left border of the left ventricle, there was calcification of the left coronary artery and portions of the circumflex and anterior descending branches (Fig. 6). There was also evidence of calcification of the aortic valve, noted on fluoroscopy. There were signs of pulmonary emphysema. The electrocardiogram appeared normal. Urinalysis, blood studies and serologic test were normal.

Diagnosis: Asymptomatic generalized arteriosclerosis, chronic bronchitis with emphysema.

Comment. Calcification of the left coronary artery, aortic valve and aortic arch was demonstrable roentgenographically and fluoroscopically. However, there was neither clinical nor electrocardiographic evidence of coronary disease despite the nature of these findings and the advanced age of the patient.

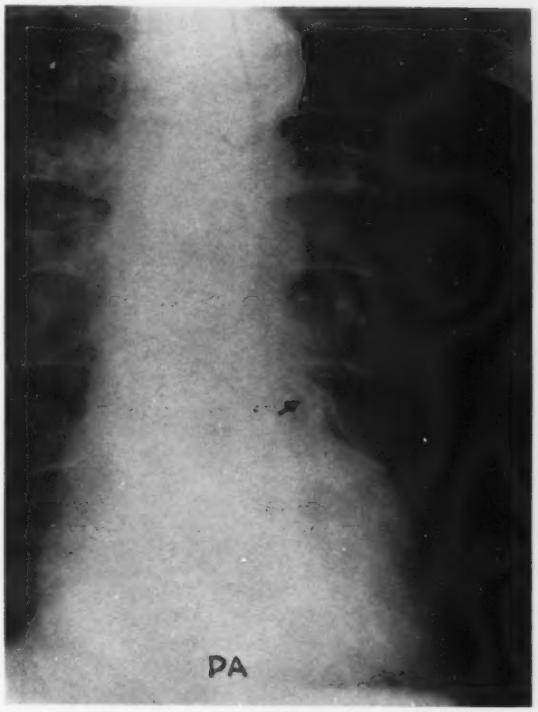

Fig. 6. Case v. McA. Posteroanterior roentgenogram showing calcification of the left coronary artery with portions of the circumflex and anterior descending branches. Aortic arch also calcified.

Case vi. A.L.V., a white female nurse sixty-five years old, suffered a stroke two years previously, and had residual weakness and incoordination of the left extremities. She complained of exertional dyspnea only. There was a history of heart disease. Examination revealed generalized angiosclerosis of the retinal arteries. The heart showed no enlargement, thrills, rubs or murmurs. There was regular sinus rhythm with a rate of 84 per minute. The second aortic sound was markedly accentuated. There was no evidence of myocardial insufficiency. Blood pressure was 210/122. There were mild residuals of a left hemiplegia.

Laboratory studies: Roentgenograms of the chest revealed a very slight generalized cardiac enlargement. There was linear calcific streaking near the upper left contour and posterior aspect of the heart at the site of the circumflex branch of the left coronary artery (Fig. 7). The aorta showed slight tortuosity. The electrocardiogram was normal, except for occasional premature contractions. Blood studies and serologic test were normal. The urine revealed a few red blood cells.

Diagnosis: Hypertensive vascular disease,

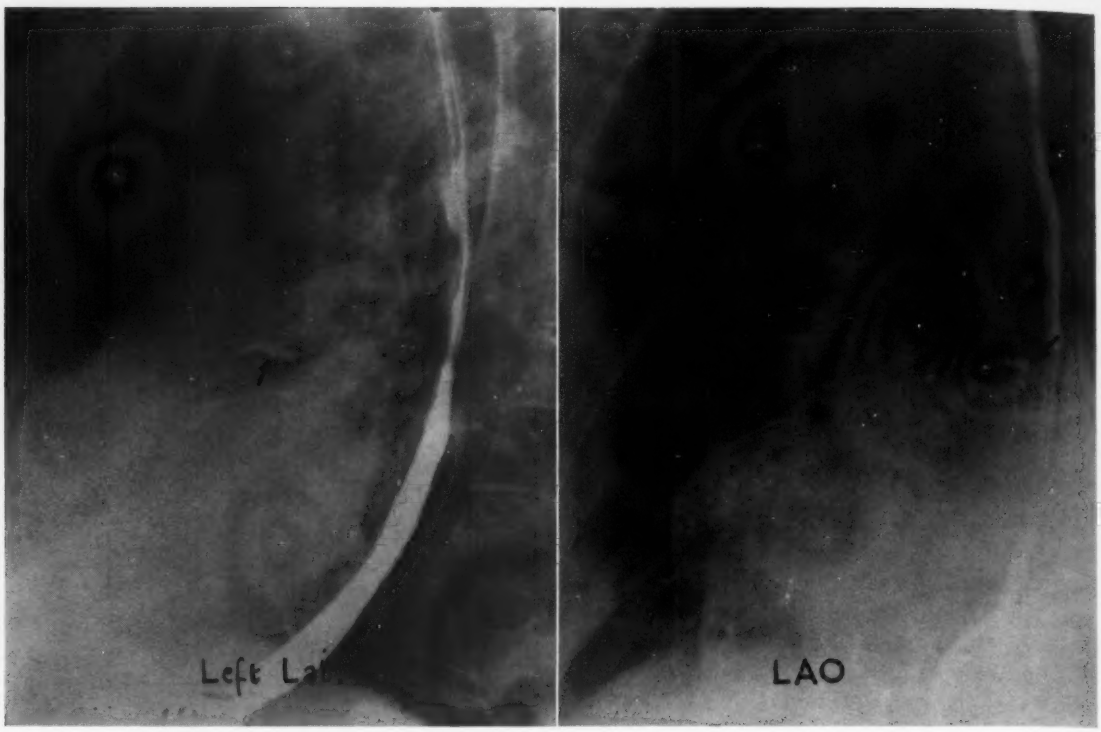

FIG. 7. Case VI. A. L. V. Left lateral and left anterior oblique views showing calcification of the circumflex branch of the left coronary artery in the auriculoventricular groove.

generalized arteriosclerosis, and mild residuals of left hemiplegia.

Comment. This patient revealed no electrocardiographic evidence of coronary artery involvement despite marked sclerotic changes on roentgenologic examination. Peripheral arteriosclerosis was manifested clinically only in the cerebral arteries.

SUMMARY

Calcification of segments of the left coronary artery was demonstrable on the chest roentgenograms of 6 patients. There were 5 males and 1 female. The average age was sixty-five years. Despite the advanced sclerotic changes in the coronary arteries, it is remarkable that 5 out of the 6 patients presented normal electrocardiograms. Only 1 patient had suffered an episode of coronary occlusion. Hypertension was present in 4 of these patients. Cardiac symptomatology was marked in only 2 patients. Clinical evidence of atheromatous degeneration was commonly manifested in the cerebral vessels and other peripheral arteries.

CONCLUSION

Calcification of the coronary arteries may be discernible on ordinary roentgenograms. The common sites and characteristics have been discussed. In our series, this finding was not associated with clinical or electrocardiographic evidence of myocardial damage or coronary insufficiency in 4 out of 6 cases. Only 1 patient presented definitive evidence of an old coronary occlusion.

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Vol. 77, No. 2

CALCIFICATION OF THE ASCENDING AORTA*

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CALCIFICATION of the ascending aorta has been set forth as a reliable sign of syphilitic aortitis. Similar calcification of nonsyphilitic origin is regarded as rare. Inasmuch as 4 of the 20 cases of calcification of the ascending aorta reported here disclosed no evidence of syphilis, neither clinically nor at necropsy, further consideration of this condition may prove informative.

Jackman and Lubert[3] reported calcification of the ascending aorta in 15 of 66 cases of syphilitic aortitis, excluding patients with aneurysms. Later, Jackman[2] observed 20 additional cases of ascending aortic calcification with positive reactions for syphilis in 15. Leighton[4] recorded ascending aortic calcification in 11 out of 20 verified cases of syphilitic aortitis, 2 of which had aneurysms. In 39 others with only clinical evidence of syphilis, 21 had ascending aortic calcifications. Thorner, Carter and Griffith[8] reported that 22 out of 122 cases of syphilitic aortitis had calcification of the ascending aorta. Of 95 cases diagnosed as syphilitic aortitis, including 30 with aneurysms, Wolkin[9] observed visible calcification of the ascending aorta in 54.

Calcification of the ascending aorta unassociated with syphilis has been observed far less frequently. Jackman and Lubert[3] found but 2 such instances in 62 cases of severe arteriosclerosis. Leighton[4] reviewed 400 consecutive chest roentgenograms unselected except for age and sex to correspond with his cases of syphilitic aortitis, and noted ascending aortic calcification in but 2, an incidence of 0.5 per cent. Thorner, Carter and Griffith[8] found only 2 in 100 cases of arteriosclerosis. Wolkin[9] encountered but 1 case in his series of 54 patients with calcification of the ascending aorta in which this calcification was not unequivocally syphilitic.

Syphilitic aortitis without aneurysm formation is often referred to as an uncomplicated syphilitic aortitis. This follows spirochetal invasion of the adventitia early in the infection, the initial lesions appearing in the proximal portion of the ascending aorta. These extend proximally and distally, and enter the media by way of the lymphatics and the vasa vasorum. The intima becomes thickened, wrinkled, and disrupted as the destruction of the medial elastic and connective tissues progress. Irregular furrows develop along the long axis of the aorta, interspersed with pearly patches of varying sizes. At first the intimal thickening may be poorly delimited, but later the margins become sharply defined and coalescent. Ulceration and calcification, which may extend into the media, are degenerative changes which follow intimal and medial necrosis.

Uncomplicated syphilitic aortitis involves principally the ascending aorta. This may be attributed to the local rich supply of vasa vasorum and lymphatics, affording an avenue for syphilitic extension greater than that for the abdominal aorta or main arteries.

As a result of syphilitic mesaortitis, the ascending aorta loses its elasticity and becomes dilated. Unless there are concomitant changes in the aortic valves, the heart is unchanged. This dilatation of the ascending aorta affords practically the only diagnostic sign of uncomplicated syphilitic aortitis. Aneurysm formation is frequent, appearing in from 10 to 20 per cent of cases. Calcification of the ascending aorta in uncomplicated syphilitic aortitis, as well as in aneurysms, occurs frequently, and is regarded as a nonspecific attempt at healing rather than as a manifestation of syphilis.

Arteriosclerosis of the aorta originates with intimal thickening, particularly about

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the origins of the direct branches such as the intercostal arteries, the carotid and the iliac arteries. The ascending aorta is involved less frequently and less extensively than the transverse and descending thoracic portions of the aorta. The intimal plaques become necrotic and softened, undergo fatty degeneration and assume an opaque, yellowish, and fairly hard consistency. As degeneration progresses, the hardened areas soften and become grumous, with erosion and ulceration of their surfaces. A surrounding cellular infiltration appears, followed by scarring. Calcification of the intima and the underlying media is one of the reparative mechanisms. Sometimes it is quite difficult to differentiate these areas from syphilitic aortitis.

As scarring becomes more pronounced, the elasticity of the aorta diminishes and varying degrees of dilatation appear. This process is most marked in the transverse, descending, and abdominal portions of the aorta. One of the reasons for the relatively uncommon localization in the ascending aorta may be that this region has no emerging branches.

Dilatation of the ascending aorta may be identified roentgenologically on postero-anterior, left anterior oblique and left lateral roentgenograms, and on angiocardiograms. The extent of pulsation may be observed fluoroscopically and can be recorded on roentgenkymograms. Steinberg and his co-workers[7] noted that the average normal diameter of the ascending aorta in 100 cases varied between 16 and 38 mm., with an average of 28.6 mm. In 51 cases of syphilis, the diameter of the ascending aorta varied between 38 and 70 mm., averaging 45.4 mm.

While dilatation of the ascending aorta is relatively common in syphilitic aortitis, it is not limited to this condition. It may also be encountered in patients with hypertension, rheumatic aortic valvular insufficiency and coarctation of the aorta. Ruffin, Castleman and White[6] described arteriosclerotic aneurysms and senile ectasia of the ascending aorta, and pointed out that large thoracic aneurysms may be arteriosclerotic or senile in nature. They regarded the combination of old age, a negative serologic reaction, and occurrence in females as suggestive of senile ectasia or arteriosclerotic aneurysms.

This overlapping of pathologic processes makes it difficult to establish definite criteria for the roentgenographic diagnosis of syphilitic aortitis. While there is no doubt that a great many patients with ascending aortic calcifications have syphilis, such calcifications alone cannot be considered absolutely indicative of pre-existent syphilis.

It is proposed for the purposes of this discussion to regard patients with a known history of syphilis or positive serologic reactions, together with calcifications of the ascending aorta, as having syphilitic aortitis. Those with calcification of the ascending aorta alone, with negative serologic reactions and no history or clinical indications of syphilis, are regarded as possibly syphilitic in origin. However, if they are over sixty years of age, they may fall into the group of senile ectasia with calcification. Considered as nonsyphilitic are those with necropsy verification in which the pathologic appearance of the aorta, as well as microscopic examination and other necropsy evidence, showed no syphilitic stigmata.

MATERIAL

Twenty cases of calcification of the ascending aorta without aneurysms were observed. Of these, 8 were definitely syphilitic, as indicated by positive serologic reactions in 7. A positive history of syphilis was obtained in 1 case, but the serologic reaction was negative. The presence of radiopaque streaks in the buttocks confirmed the fact that this patient had received antisyphilitic treatment. In one case necropsy confirmation was available. There were 8 classified as possibly syphilitic, and 4 in which neither the clinical, serologic, nor necropsy changes indicated the presence of syphilis.

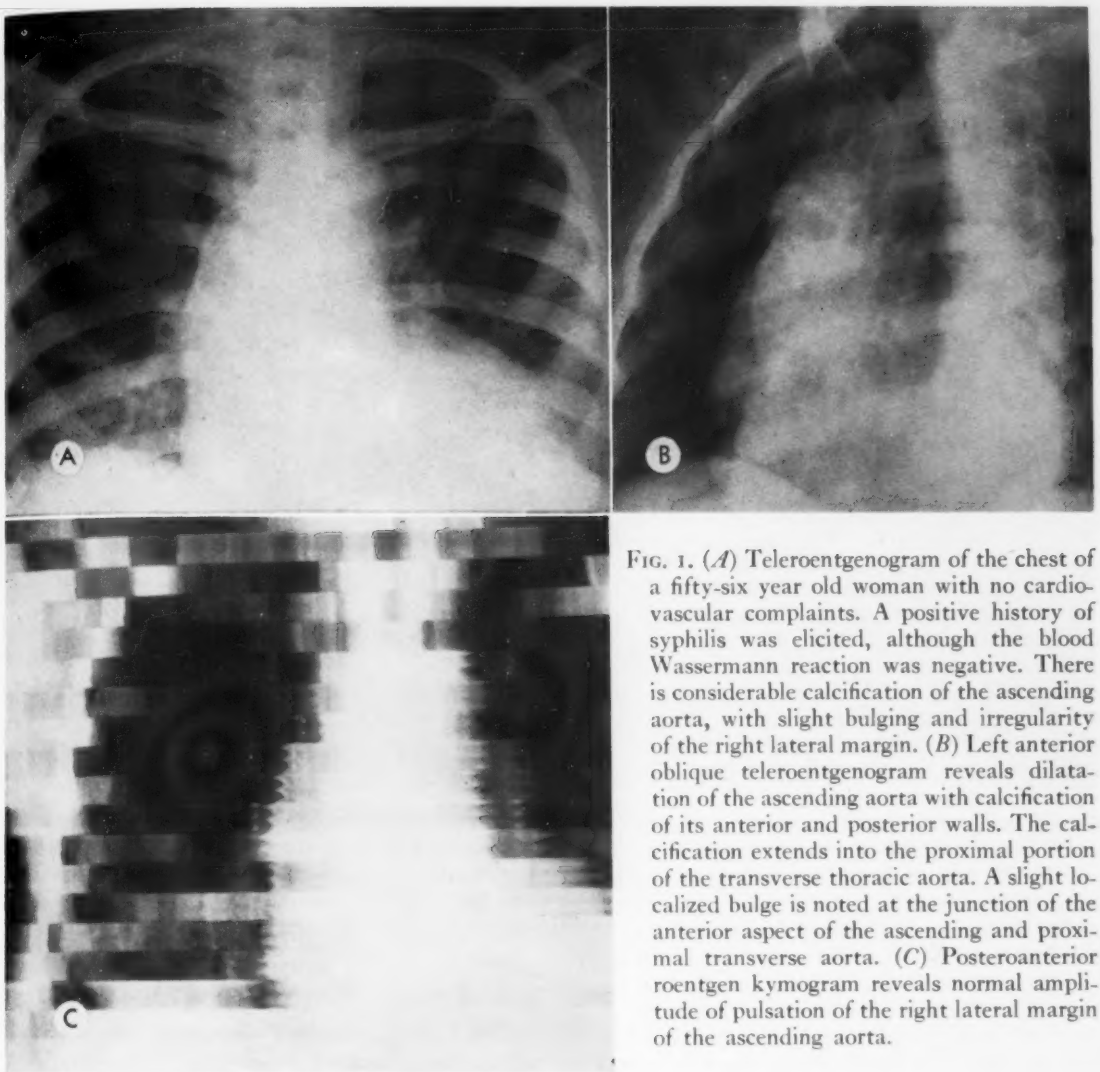

Fig. 1. (*A*) Teleroentgenogram of the chest of a fifty-six year old woman with no cardiovascular complaints. A positive history of syphilis was elicited, although the blood Wassermann reaction was negative. There is considerable calcification of the ascending aorta, with slight bulging and irregularity of the right lateral margin. (*B*) Left anterior oblique teleroentgenogram reveals dilatation of the ascending aorta with calcification of its anterior and posterior walls. The calcification extends into the proximal portion of the transverse thoracic aorta. A slight localized bulge is noted at the junction of the anterior aspect of the ascending and proximal transverse aorta. (*C*) Posteroanterior roentgen kymogram reveals normal amplitude of pulsation of the right lateral margin of the ascending aorta.

Syphilitic Group. Four of the 8 patients in this group were males. The age incidence varied from forty-five to seventy years, half being less than sixty years. One patient had calcified aortic stenosis, verified at necropsy.

Chest roentgenograms were available in all 8 cases. The calcification in the ascending aorta was considered extensive in 5, moderate in 1, and in 1 the calcification was represented only by a thin linear streak on the right anterolateral wall of the aorta. Those with extensive calcifications presented segmental lime salt deposits, the plaques varied considerably in size and merged with one another frequently. The diameter of the ascending aorta, measured on left anterior oblique or left lateral roentgenograms approximately at its midpoint, varied from 3.5 to 5.5 cm. When the calcification encircled the entire aorta, the diameter of the ascending aorta could be ascertained on overpenetrated frontal films as well. In 5 cases the ascending aorta measured from 5.0 to 5.5 cm. in diameter. In one the diameter was 4.5 cm., and in the remaining 2 it was between 3.6 and 4.0 cm. In 5 cases the thickness of the wall of the ascending aorta could be measured. This varied from 3 to 6 mm., including the

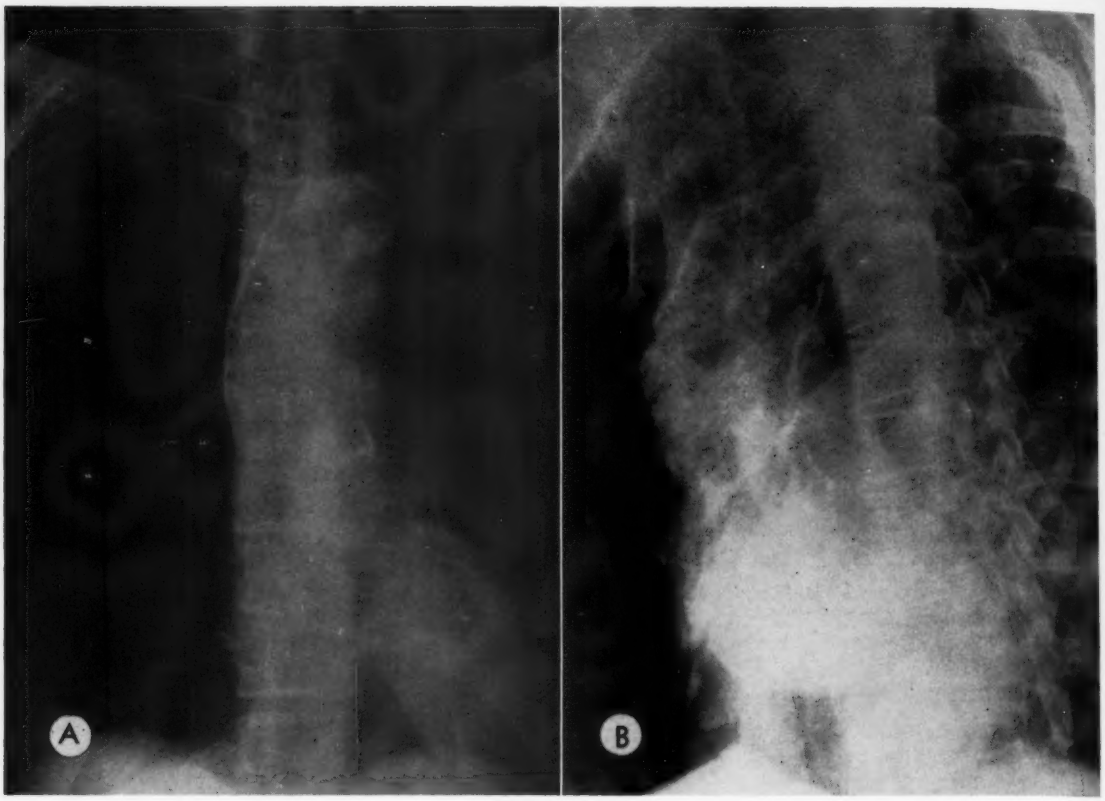

Fig. 2. (*A*) Overpenetrated posteroanterior teleroentgenogram of a fifty-seven year old man with a positive reaction for syphilis in the blood. As seen in the frontal projection considerable calcification of the ascending aorta extending into the transverse thoracic aorta is present. The right border of the aorta is not conspicuous. However, the diameter of the aorta can be seen to be dilated because the medial border is visible to the left of the mid-thoracic spine. (*B*) Left anterior oblique roentgenogram reveals diffuse confluent calcification of the dilated ascending aorta.

calcific layer, which in itself was from 1 to somewhat less than 3 mm. in thickness in the various patients.

Calcifications were visible in the transverse thoracic aorta in 7 of the 8 cases. In 4 the calcification was moderate, and in the remaining 3 it was extensive. The aortic knob as seen on posteroanterior teleroentgenograms was considered normal in 1 patient, slightly prominent in 4, and moderately prominent in 3. The diameter of the transverse thoracic aorta measured on left anterior oblique or left lateral teleroentgenograms varied from 3.5 to 4.0 cm.

Calcification of the descending thoracic aorta was observed in 4 cases. These were estimated as slight in 1, as moderate in 2, and as advanced in 1. No aneurysm of the descending thoracic aorta was identified in this series, the presence of such dilatation serving to eliminate the case from the present study.

Probable Syphilitic Calcification of the Ascending Aorta. This group included 8 patients varying in age from fifty-eight to eighty-one years. Five were males. Negative serologic reactions for syphilis were obtained in all, and in none was there any clinical suggestion of syphilis. No valvular disease was present. Chest roentgenograms were available in all. The calcification of the ascending aorta was advanced in 6, and mild in the remaining 2. The lime salt deposits were segmental in those patients with mild calcification. When advanced,

the deposits were segmental, but often the plaques merged so that in 3 the ascending aorta was practically a uniformly calcified tube. The diameter of the ascending aorta was less than 4.0 cm. in 5 patients. In 2 it was from 4.5 to 5.0 cm. in diameter, and in 1 the diameter was 6.0 cm. The thickness of the aortic wall measured from the inner aspect of the calcification to the outer soft tissue shadow of the ascending aorta in 4 cases varied from 2.5 mm. to 4.0 mm. The calcifications themselves measured from 1.0 to 2.5 mm. in depth.

Calcification of the aortic arch of pronounced degree was noted in 3 of these patients, 1 of whom had only moderate calcification of the ascending aorta. In 4 cases calcification of the transverse thoracic aorta was only moderate, and in 2 no calcification in this region was visible. The diameter of the arch of the aorta was below 3.5 cm. in 5 cases, and up to 4.0 cm. in the remaining 3. The aortic knob was considered to be moderately prominent in 2 cases. In 5 there was a slight degree of prominence, while in 1 the shadow was entirely within normal limits.

Calcification of the descending thoracic aorta of an advanced form was present in 2 patients. In the remaining 6 very little such calcification was observed.

Comment. It is difficult to make a definite diagnosis of syphilitic aortitis in these patients, the only change pointing toward a syphilitic origin being calcification of the ascending aorta.

Against this is the absence of a positive history of syphilis, and negative serologic and clinical evidence of syphilis. The absence of dilatation or only slight dilatation of the ascending aorta also points away from the possibility of a syphilitic origin for the ascending aortic calcification in this group. It is realized that a negative serologic reaction for syphilis is by no means uncommon with aortitis. Perhaps additional serologic investigations, such as the Treponema immobilization test, will help clarify this question.[1]

Fig. 3. Left anterior oblique roentgenogram of a seventy year old male patient with no cardiovascular complaints but with a positive history of syphilis. There is extensive calcification of the entire aorta, extending from the aortic valves to the distal descending thoracic aorta. No localized aneurysmal dilatation is visible.

Nonsyphilitic Calcification of the Ascending Aorta. There were 4 patients in this group, all between sixty and seventy years of age. Two were males. In 1, a female sixty-two years of age, calcareous aortic stenosis was present. All of these individuals had chest roentgenograms, and the observations were confirmed at necropsy. In the patient with aortic valve disease, the heart weighed 200 grams, and it was the opinion of the pathologist (Dr. David Grayzel) that the lesion was of rheumatic origin. The causes of death in these 4 respectively were leukemia, carcinoma of the rectum, cerebral embolus, and thrombosis of the subclavian artery following operation for carcinoma of the rectum. The serologic examinations for syphilis were negative in 3 of the 4 cases, while in the fourth the test had not been performed.

Fig. 4. Left anterior oblique roentgenogram of a seventy-eight year old woman with mild hypertention, but neither clinical nor serologic indications of syphilis. Note the extensive calcification of the ascending and transverse thoracic aorta. Only mild dilatation of the ascending aorta is present.

Postmortem examinations disclosed no luetic stigmata (Dr. David Grayzel, 3 cases; Dr. James Berkman, 1 case). The appearance of the ascending aorta in these individuals was one of extensive atherosclerosis with calcification. No tree-barking could be made out, and microscopic examination of the tissues of the ascending aorta failed to reveal any mesaortitis. While it is conceivable that syphilis had been present, one would have to presume that such changes had been completely eradicated, and there certainly was no evidence either in the history, in the physical examinations, or at necropsy to support this supposition.

Advanced calcification of the ascending aorta was noted in 2 cases, while in the other 2 the calcification was moderate. In the 2 cases with advanced calcification the lime salt deposits were of a segmental nature with confluence of the sclerotic plaques so extensive that the ascending aorta was rendered into a rigid tube. In the first case no calcification of the aortic arch or the descending thoracic aorta was seen, while in the second the calcification extended to involve the transverse and descending aorta. In the 2 cases with moderate calcification of the aorta, 1 had moderate calcification of the arch with no calcification in the descending thoracic aorta. The fourth had neither calcification of the arch nor of the descending thoracic aorta.

The diameter of the ascending aorta in all 4 patients was 4.0 cm. or less. In 3 the aortic knob was slightly prominent. The diameter of the transverse thoracic aorta was 4.0 cm. or less in all 4.

Roentgen Kymographic Examinations. Kymograms of the heart and great vessels were made in the posteroanterior and left anterior oblique projections in 4 patients with syphilitic aortitis. In 3 of these, the calcification of the ascending aorta was

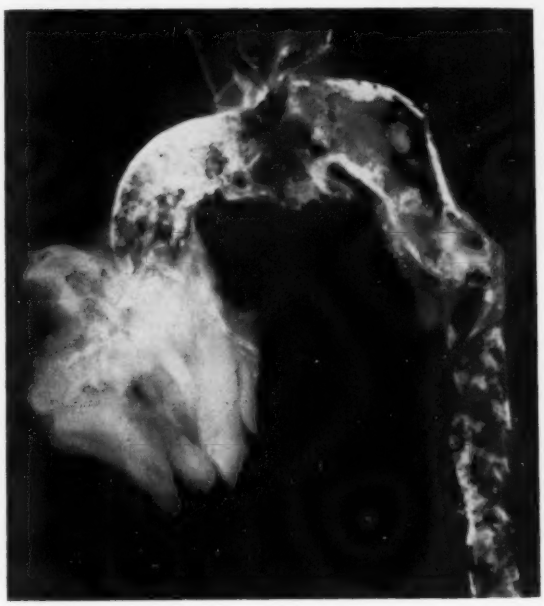

Fig. 5. Postmortem roentgenogram of the heart and aorta of an eighty-one year old woman with no cardiovascular disturbance. There were no clinical nor serologic evidences of syphilis, nor were syphilitic changes encountered at postmortem examination (Dr. James Berkman). Note the extensive calcification of the entire aorta, most pronounced in the ascending and transverse thoracic portions. Advanced calcification of the coronary arteries is present.

extensive and in 1 moderate. In 1 patient extensive calcification of the transverse thoracic aorta was present, together with moderate calcification of the descending thoracic aorta. In the remaining 3 patients, the aortic knob showed only mild calcification.

The roentgen kymograms in all 4 patients showed definite pulsation of the ascending aorta and of the aortic knob. The movement of the aortic walls in 1 patient with extensive calcification was synchronous with that of the left ventricle. At necropsy this patient was found to have an infarct involving the anterolateral aspect of the left ventricle. In this case it could not be determined whether the pulsations of the ascending aorta were expansile or transmitted. The movement of the aorta was opposite in phase to that of the left ventricle in the other 3 patients. By checking on the movements of the opposite walls of the aorta, particularly as seen in the left anterior oblique view, it was possible to observe the movement of the anterior and posterior walls in 2 patients. In 1 the opposite walls moved in opposite directions, so that the pulsations were considered expansile. In the other the movement of the anterior and posterior walls was in the same direction at the same instant, so that the pulsation was considered transmitted, and probably reflected the impulses produced by the entrance of blood into the aorta during ventricular systole.

Comment. Maynard[5] pointed out that for the roentgenologic diagnosis of syphilitic aortitis one required undoubted evidence that the patient had syphilis, that any other disease which might cause aortic dilatation was absent, and that the patient was younger than forty years of age. Dilatation of the aorta as demonstrated roentgenologically was considered an important sign. A hollow accentuated aortic second sound and possibly a systolic murmur at the aortic area were also considered diagnostic. Steinberg and his co-workers[7] investigated the angiocardiographic aspects of syphilitic aortitis, and concluded that dilatation of the mid-ascending aorta beyond 38 mm. was strong confirmatory evidence. They mentioned that irregularity of the lumen of the ascending aorta, variation in its thickness and tortuosity of the aorta all might be observed with syphilitic aortitis. In the 51 syphilitic cases the diameter of the ascending aorta varied from 38 to 70 mm., with an average of 45.4 mm. These authors commented that abnormal dilatation of the ascending aorta might be observed with hypertension, rheumatic aortic valvular insufficiency, and coarctation of the aorta. They did not believe that aortic dilatation above the upper limit of normal could be attributed to arteriosclerosis alone. The coexistence of hypertension and syphilis occasionally complicated the interpretation of dilatation of the ascending aorta. However, in their opinion, the absence of aortic dilatation did not in itself exclude syphilis. Included among their criteria was calcification of the ascending aorta, which was present in 26.6 per cent of their 51 cases.

In reviewing the data presented here, 8 of the 20 patients undoubtedly had syphilis. Eight had neither clinical nor serologic evidence of syphilis but presented calcification of the ascending aorta. Four others had calcification of the ascending aorta with neither clinical, roentgenologic nor necropsy evidence of pre-existent syphilis. The last 4 are of particular interest inasmuch as they indicate that calcification of the ascending aorta may exist without demonstrable evidence of syphilis. In the group with syphilis, dilatation of the ascending aorta was common. In the doubtful group of 8 patients dilatation of the ascending aorta was observed in 3 patients, while in 5 no such definite dilatation could be observed. The diameter of the ascending aorta in all 4 patients with no evidence of syphilis was normal.

CONCLUSION

Inasmuch as it is well recognized that calcification of the ascending aorta is not in itself a manifestation of syphilitic infection, the evidence presented here suggests

that infrequently calcification of the ascending aorta may be of nonsyphilitic origin. Should the diameter of the ascending aorta, particularly in elderly patients, be within normal limits, the possibility of a nonsyphilitic origin has to be considered. Should the diameter of the ascending aorta be above normal, a syphilitic origin is more likely.

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PHLEBOGRAPHIC STUDY OF THE SUPERIOR VENA CAVA*

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THE anatomic study of the vascular structures of the mediastinum is successfully carried out by angiocardiography. Phlebography is a useful procedure to study the pathology of the superior vena cava and the formation of a collateral circulation produced by pulmonary and mediastinal pathologic processes.

FIG. 1. Normal superior vena cava. Notice its position in the mediastinum and the uniformity of its contours.

FIG. 2. Normal superior vena cava in a patient with far advanced pulmonary tuberculosis and chronic cor pulmonale.

Conventional roentgen examination of the chest gives only limited information regarding the superior vena cava in normal and pathologic states, while mediastinal phlebography and angiocardiography are useful aids in determining the site and nature of the obstruction, as well as the pattern of the collateral circulation. Roswit, Kaplan and Jacobson[7] mention phlebography as being of value in the diagnosis of the superior vena cava obstruction syndrome in cases of bronchogenic carcinoma, syphilitic aortic aneurysms, malignant lymphomas, mediastinal fibrosarcomas, etc. These authors state that interruption of blood flow in the superior vena cava is accomplished by the primary bronchial neoplasm or invaded lymph nodes, mainly through external compression, occasionally by intraluminal invasion and rarely by

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FIG. 3. Superior vena cava in a left fibrothorax. It is pulled into the left hemithorax and is bent at the point of emergence from the innominate vein.

actual thrombosis. As soon as the major venous trunks or their tributaries become affected, there develops a wide collateral circulation. The interesting features of this collateral circulation were described in detail by McIntire and Sykes.[5]

According to Roswit, Kaplan and Jacobson[7] clinical evidence of superior vena cava obstruction is often absent. In their series of 38 patients, angiography was performed before and after radiation therapy in 8 patients and in all proved to be of value. The majority of their patients, however, when first seen were so seriously ill that even this relatively simple and safe diagnostic procedure was contraindicated.

Because of the rapidly rising incidence of bronchogenic carcinoma, the relative frequency of obstruction of the superior vena cava in the natural history of this disease, and the marked benefits which may be derived from prompt treatment, phlebography is becoming of increasing imporance in the study of the superior vena cava.

Jordan, *et al.*,[2] report a case of unsuspected superior vena cava obstruction detected by means of angiocardiography. Lindblom,[4] and Gvozdanović and Oberhofer[1] stress the importance of mediastinal phlebography in the diagnosis of mediastinal tumors and syphilitic aortic aneurysms. Roberts, Dotter and Steinberg,[6] after a study of 1,500 angiograms, add much to our knowledge concerning the superior vena cava in the normal and pathologic states. They mention the congenital alterations which can be detected by angiocardiography and point out that 78 per cent of the cases with superior vena cava obstruction are due to malignant tumors. Salén[8] reports 3 cases of superior vena cava obstruction and

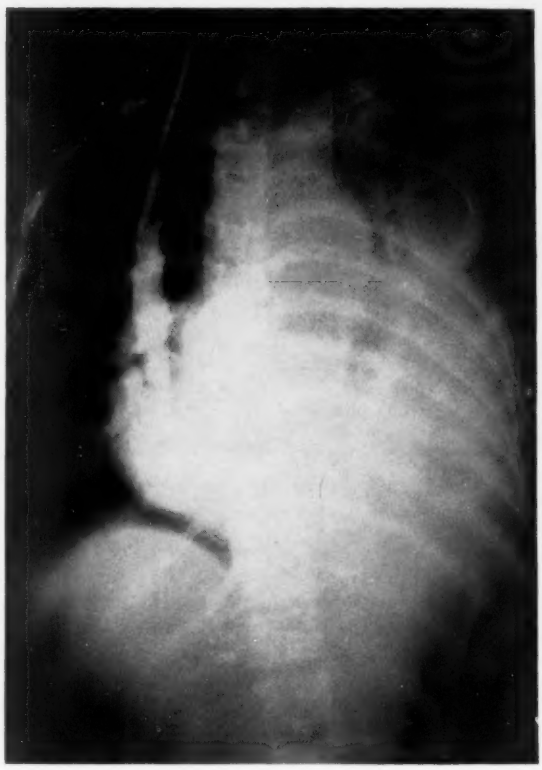

FIG. 4. Superior vena cava in a left-sided malignant mesothelioma. The superior vena cava is displaced to the right.

points out that a collateral circulation is established through the thoracic wall veins, the azygos system and internal mammary veins. Kjellberg and Olsson[3] were able by means of phlebography to detect a sphincter mechanism at the point of junction of the superior vena cava and pulmonary veins with the auricles. In a recent report, Steinberg[9] refers to a polypoid type of invasion of the superior vena cava in bronchogenic carcinoma cases, a sign which indicates inoperability.

The purpose of this paper is to call attention to the most important types of alteration shown by means of phlebography in the superior vena cava, not only in demonstrating malignant neoplasms, but also from the anatomic point of view. Our study is based on a review of 1,224 angiograms.

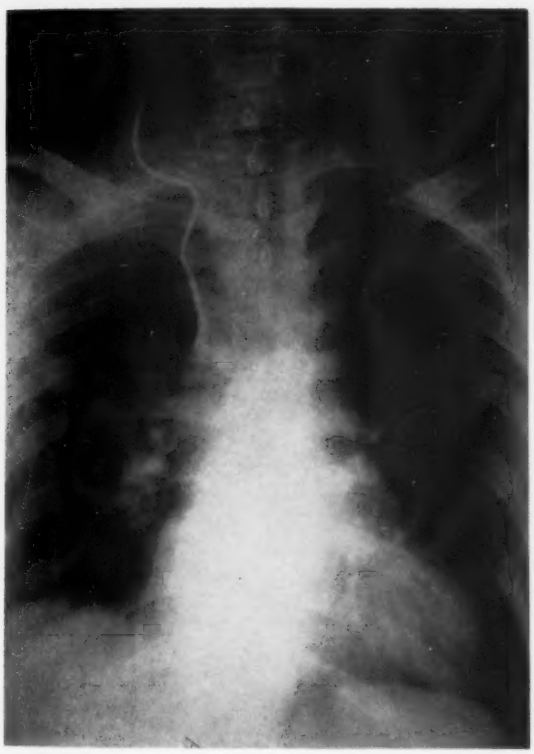

FIG. 5. Complete stenosis of the superior vena cava in a case of mediastinal Hodgkin's disease following prolonged radiation therapy. The tip of the catheter is seen at the level of the pulmonary hilus.

FIG. 6. Same case as Figure 5. The contrast medium was injected through the external jugular vein. There is wide collateral circulation through the prevertebral and intercostal veins. The superior vena cava is reduced to a very narrow channel in its upper portion and is completely stenosed in its lower portion.

FINDINGS

Changes in the Position of the Superior Vena Cava. The superior vena cava follows the mediastinum in its position. Any changes in the position of the mediastinum due to pulmonary or extrapulmonary alterations will change the position of the superior vena cava. The normal superior vena cava as detected by phlebography is situated at the right border of the vertebral column (Fig. 1 and 2). In cases with pulmonary fibrosis or extensive pachypleuritis (fibrothorax, organized empyema, old pneumothorax or hemothorax), the superior vena cava is deviated towards the affected side, and in cases with disease on the left side, it can be seen on the left side of the vertebral column. In some cases,

FIG. 7. Upper mediastinal tumor (aneurysm of the arch of the aorta) compressing the right innominate vein. There is a wide collateral circulation in the shoulder. The superior vena cava is bent and displaced to the left.

the superior vena cava can be seen bending in its upper portion and close to the point of emergence from the innominate vein (Fig. 3). When there is a pathologic process which displaces the mediastinum into the right hemithorax, the superior vena cava is found to be clearly on the right side, as was the case in a diffuse mesothelioma of the pleura in which the mediastinum was found to be deviated to the right (Fig. 4). These changes in the position of the superior vena cava do not affect its anatomic integrity, reflecting only changes in its topography.

Obstructive Processes of the Superior Vena Cava. Among those pathologic processes which affect the superior vena cava by compression, invasion or collapse of its walls, the most important are:

1. Formation of a scar tissue following prolonged radiation therapy. Complete stenosis of the superior vena cava after roentgen therapy in cases of Hodgkin's disease, lymphoma or lymphosarcoma have been reported (Fig. 5 and 6). The pathogenesis of this process is not clear. It may well be that the fibrous scar tissue which has developed in the mediastinum compresses the superior vena cava, or that the fibrous process has affected the walls of the vein itself.

2. Compression of the superior vena cava due to tumors of the superior mediastinum (aneurysm, dermoid cyst, lymphosarcoma) and rarely by lymph node enlargement. This compression of the superior vena cava is usually slow and progressive, permitting the formation of a wide collateral circulation. This varies according to the site of compression. If it is in the upper portion of the superior vena cava (compression of the innominate vein), the collateral circulation is unilateral and

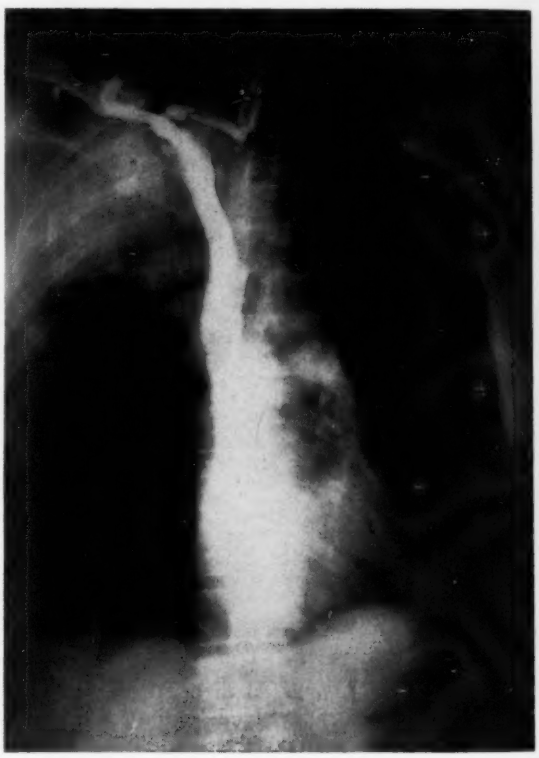

FIG. 8. Bronchogenic carcinoma of the upper right bronchus (Pancoast type). The superior vena cava is slightly narrower than normal.

localized in the shoulder and upper portion of the corresponding hemithorax (Fig. 7). If the obstruction is localized above the azygos vein, the collateral circulation will, in most cases, be bilateral, and if the obstruction is located below the azygos vein, the collateral circulation will be both thoracic and abdominal.

3. Invasive processes of the superior vena cava. Most important of these is bronchogenic carcinoma, mainly of the right upper lobe bronchus. In an early stage and when the pleura is not invaded, it is possible to find a normal superior vena cava; in more advanced stages the carcinoma may invade and stenose the vessel, either through direct extension or by compression by the primary neoplasm or the metastatic lymph nodes.

In most cases of bronchogenic carcinoma arising in the upper lobe bronchus, the superior vena cava is diminished in its caliber just above the upper lobe bronchus (Fig. 8). This finding indicates inoperability. In more advanced cases, the stenosis may be cylindric, involving a larger portion of the superior vena cava (Fig. 9). Sometimes the stenosis is found to be concentric, in an hour glass fashion (Fig. 10) even though the roentgen appearance may be that of a relatively recent cancer. This type of stenosis has been encountered in cases of very malignant and invasive neoplasms.

Fig. 9. Cylindric type of stenosis of the superior vena cava in a right upper lobe bronchogenic carcinoma.

Fig. 10. Infundibular type of stenosis of the superior vena cava due to a carcinoma of the right main bronchus. Note that the external jugular vein is markedly dilated.

In other cases the bronchogenic tumor protrudes outwards and invades the superior vena cava close to the upper lobe bronchus. In these instances a polypoid defect will be shown in the superior vena cava, just above the involved bronchus (Fig. 11).

In all of these cases surgery is not indicated. We think that these signs, demonstrated somewhat early in the course of the

FIG. 11. Polypoid invasion of the superior vena cava (adenocarcinoma of the right upper lobe bronchus). Thoracotomy performed previously had shown an extensive mediastinal lymph node invasion.

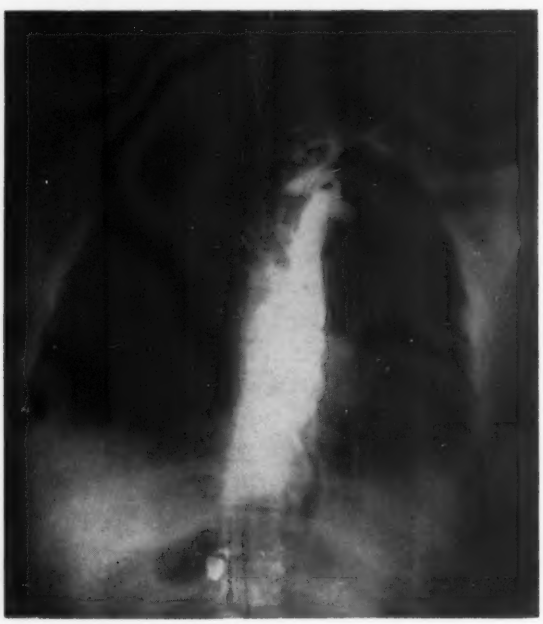

FIG. 12. Complete obstruction of the superior vena cava probably due to thrombosis. The left common trunk of the superior intercostal veins is opacified.

FIG. 13. Same case as Figure 12, lateral view. The lateral phlebogram shows the intercostal veins branching off a common trunk and absence of the superior vena cava.

disease, are of the greatest importance.

4. Intrinsic obstruction of the superior vena cava is rare; however, cases have been reported caused by thrombosis of the superior vena cava. This was the case in one of our patients (Fig. 12 and 13). In some instances a thrombus in the hepatic veins may extend to the superior vena cava.

Finally, the superior vena cava may be the site of congenital alterations, the most important of which are stenosis and congenital absence.

SUMMARY

A study of 1,224 angiograms was made in order to determine what important abnormal changes, due to disease processes, occur in the superior vena cava.

Mention is made of phlebography as a useful procedure for study of the pathology of the superior vena cava and of the formation of a collateral circulation brought about by pulmonary and mediastinal pathologic processes.

Obstructive processes of the superior vena cava are frequent, the most important being those due to bronchogenic car-

cinoma (mainly of the upper right bronchus). The signs of obstruction as demonstrated by phlebography early in the course of the disease are of the greatest importance.

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THE ARTERIES OF THE ABDOMEN, PELVIS, AND THIGH*

I. NORMAL ROENTGENOGRAPHIC ANATOMY

II. COLLATERAL CIRCULATION IN OBSTRUCTIVE ARTERIAL DISEASE

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THE frequent use of translumbar aortography for the detection of renal abnormalities[8,12,33] and obstructive arterial disease[11,21,22,30,32] has stressed the need for a detailed description of the normal roentgenographic anatomy of the arteries of the abdomen and pelvis. Since no such detailed account is available, a study was undertaken of 182 technically satisfactory aortographic examinations in 173 patients performed at the University of Michigan Hospital. An attempt was made to identify every artery visualized in these aortograms. Simultaneous observations and dissections in the anatomy laboratory were of great value in the identification and localization of the majority of arteries described in standard anatomy texts.

Although numerous reports emphasize the value of aortography in delineating obstructive arterial disease, only one[18] of these includes a systematic account of the visible collateral circulation around the major sites of occlusion. This deficiency provided the incentive for an additional detailed study of the collateral circulation occurring in about 151 obstructions seen in 91 patients.

PART I. NORMAL ROENTGENOGRAPHIC ANATOMY

The characteristic position, size, and distribution of the principal arteries of the abdomen and pelvis as they usually are observed in aortograms are illustrated in Figure 1 and Figure 2. Many variations of normal occur to alter the course, caliber, and positions of these vessels. These diagrams, therefore, are a guide to the usual and are not applicable to all cases. Some of the more constant anatomic and roentgenographic findings, as well as a few of the variations of the normal which have been observed roentgenographically, are discussed below. Representative normal aortograms are illustrated in Figures 3, 4 and 5.

THE ABDOMINAL AORTIC BRANCHES

Ventral Unpaired Branches. The celiac artery characteristically arises from the aorta at L-1 and promptly divides into the splenic artery, its largest division, the hepatic artery, intermediate in size, and the left gastric artery, the smallest of the terminal branches. The splenic artery is easily recognized by its large size and markedly tortuous course in the left upper quadrant of the abdomen. It provides the dorsal pancreatic artery close to its origin, the pancreatica magna artery midway along its course, and the left gastroepiploic artery just proximal to its division into terminal branches. Several small short gastric arteries may arise from the superior terminal branch to supply the fundus of the stomach. The numerous branches of the splenic artery are all small in caliber and are visualized only infrequently. The hepatic artery, the second of the celiac divisions, can easily be traced proximally from its terminal arborizations in the liver. Close to its origin, the common hepatic provides a small right gastric artery and slightly more distally a larger gastroduodenal artery. Both of these vessels arise nearly at right angles to the hepatic artery.

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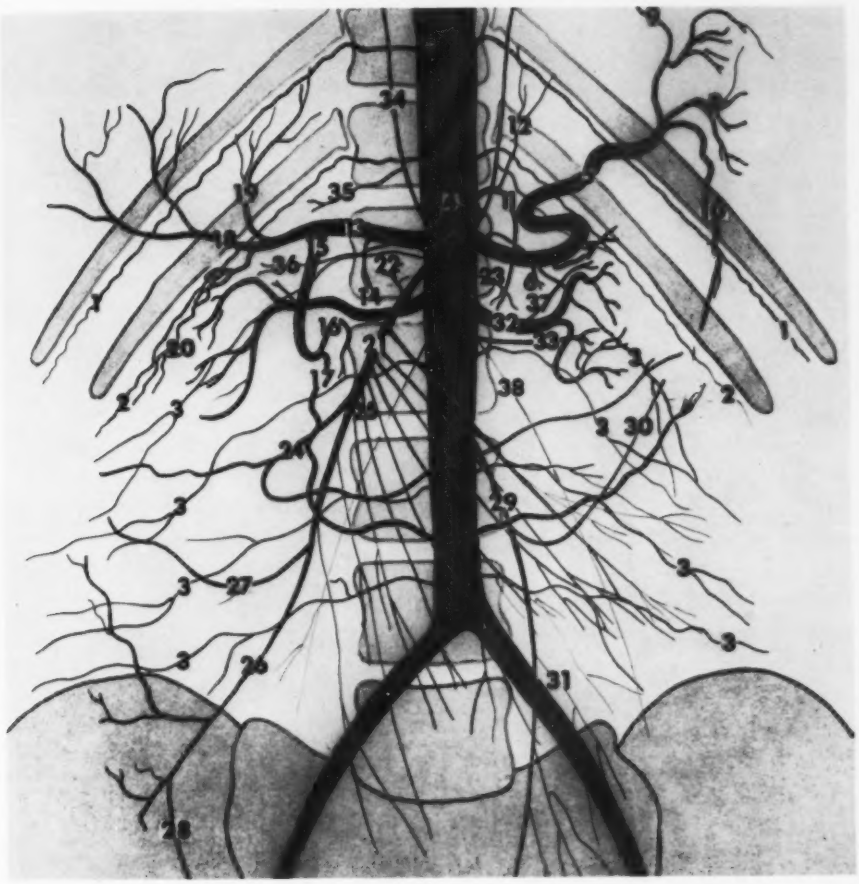

Fig. 1. The arteries of the abdomen.

No.	*Artery*
1.	intercostals
2.	subcostals
3.	lumbar
4.	celiac
5.	splenic
6.	dorsal pancreatic
7.	pancreatica magna
8.	terminal branches to spleen
9.	short gastric
10.	left gastroepiploic
11.	left gastric
12.	esophageal
13.	common hepatic
14.	right gastric
15.	gastroduodenal
16.	anterior superior pancreaticoduodenal
17.	right gastroepiploic
18.	right hepatic
19.	left hepatic
20.	cystic
21.	superior mesenteric
22.	inferior pancreaticoduodenal
23.	inferior pancreatic
24.	middle colic
25.	intestinal
26.	ileocolic
27.	right colic
28.	appendiceal
29.	inferior mesenteric
30.	left colic
31.	sigmoid
32.	renal
33.	accessory renal
34.	inferior phrenic
35.	superior suprarenal
36.	middle suprarenal
37.	inferior suprarenal
38.	internal spermatic

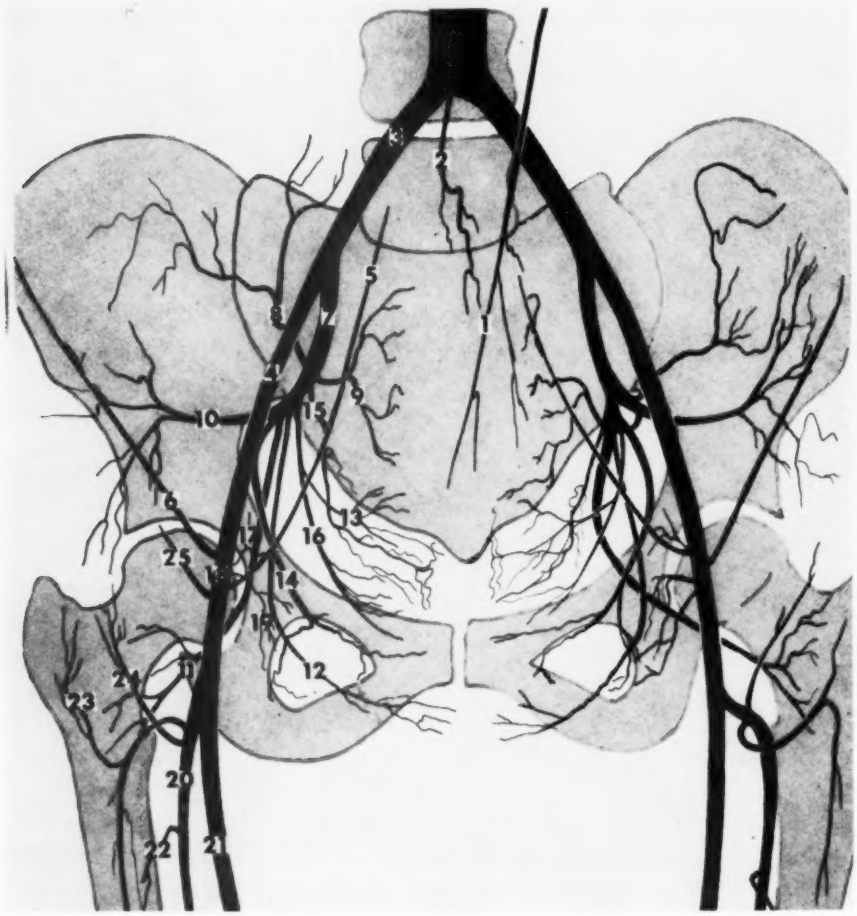

FIG. 2. The arteries of the pelvis.

No.	*Artery*
1.	superior hemorrhoidal
2.	middle sacral
3.	common iliac
4.	external iliac
5.	inferior epigastric
6.	deep circumflex iliac
7.	hypogastric
8.	iliolumbar
9.	lateral sacral
10.	superior gluteal
11.	inferior gluteal
12.	internal pudendal
13.	middle hemorrhoidal
14.	obturator
15.	uterine
16.	vesical
17.	superficial epigastric
18.	common femoral
19.	external pudendal
20.	profunda femoris
21.	superficial femoral
22.	perforators
23.	lateral femoral circumflex
24.	medial femoral circumflex
25.	superficial circumflex iliac

The right gastric artery may appear to course superiorly or inferiorly, depending upon the position of the stomach at the time of filming. Occasionally, it arises distal to the gastroduodenal artery. The gastroduodenal artery provides the posterior superior pancreaticoduodenal artery near its origin and then descends to bifurcate into its two terminal branches, the small anterior superior pancreaticoduodenal artery and the larger right gastroepiploic artery. The latter vessel courses in serpentine fashion across the abdomen along the greater curvature of the stomach

Fig. 3. Normal abdominal aortogram. The left gastric artery (1) is particularly well shown.

to meet the left gastroepiploic artery arising from the splenic artery. Terminally, the hepatic proper divides into the left and right hepatic arteries, the latter usually providing the cystic artery. The left gastric artery, the third branch of the celiac artery, is frequently difficult to identify because of its close proximity to the contrast-filled aorta and because of its inconstant position, varying of necessity with differing types and positions of the stomach. It sometimes may be seen giving origin to small esophageal arteries in the most superior portion of its arch, and, occasionally, it provides an accessory or replacing left hepatic artery.[27] The inferior pancreatic artery usually arises as a branch of the dorsal pancreatic artery,[34] but this vessel is rarely identified roentgenographically.

The superior mesenteric artery arises 1–2 cm. below the celiac origin and promptly provides the seldom-visualized inferior pancreaticoduodenal arteries. Its first major branch is the large middle colic artery, which is extremely variable in its position and often difficult to identify in the normal state. An accessory right hepatic artery, or even the common hepatic artery, may arise infrequently from the superior mesenteric artery close to its origin. Further distally, a variable number of intestinal branches originate and form a fanlike ramification covering the entire lower abdomen. The main stem of the superior mesenteric artery courses obliquely, inferiorly, and to the right from its origin to the right lower quadrant where it terminates, providing the right colic, ileocolic, and appendiceal arteries. The right colic artery is often absent.

The inferior mesenteric artery, much smaller than the superior mesenteric, originates approximately at L-3 and descends usually to the left of the aorta to terminate as the superior hemorrhoidal artery in the true pelvis. In its course it provides the readily identified left colic artery, which follows a smooth curvilinear course to the splenic flexure and an inconstant number of branches to supply the sigmoid colon.

Paired Lateral Branches. The paired lateral branches include the inferior phrenic arteries, the suprarenal arteries, the renal arteries, and the internal spermatic (ovarian) arteries. The inferior phrenic arteries appear to arise from the aorta just lateral or superior to the celiac axis and run sharply superiorly along the crura of the diaphragm to divide into two terminal branches. The suprarenal arteries are derived from three sources, the superior suprarenals from the inferior phrenic arteries, the middle suprarenals from the aorta, and the inferior suprarenals from the left and right renal arteries. These vessels are extremely variable in size and number; they frequently are impossible to identify. The renal arteries arise nearly opposite each other from the lateral portion of the aorta at the lower margin of L-1 or the body of L-2. Accessory renal arteries are commonly encountered (23 per cent[13]), more often on the left side. Immediately below the renals, the internal spermatic (ovarian) arteries are often seen. These are easily recognized by

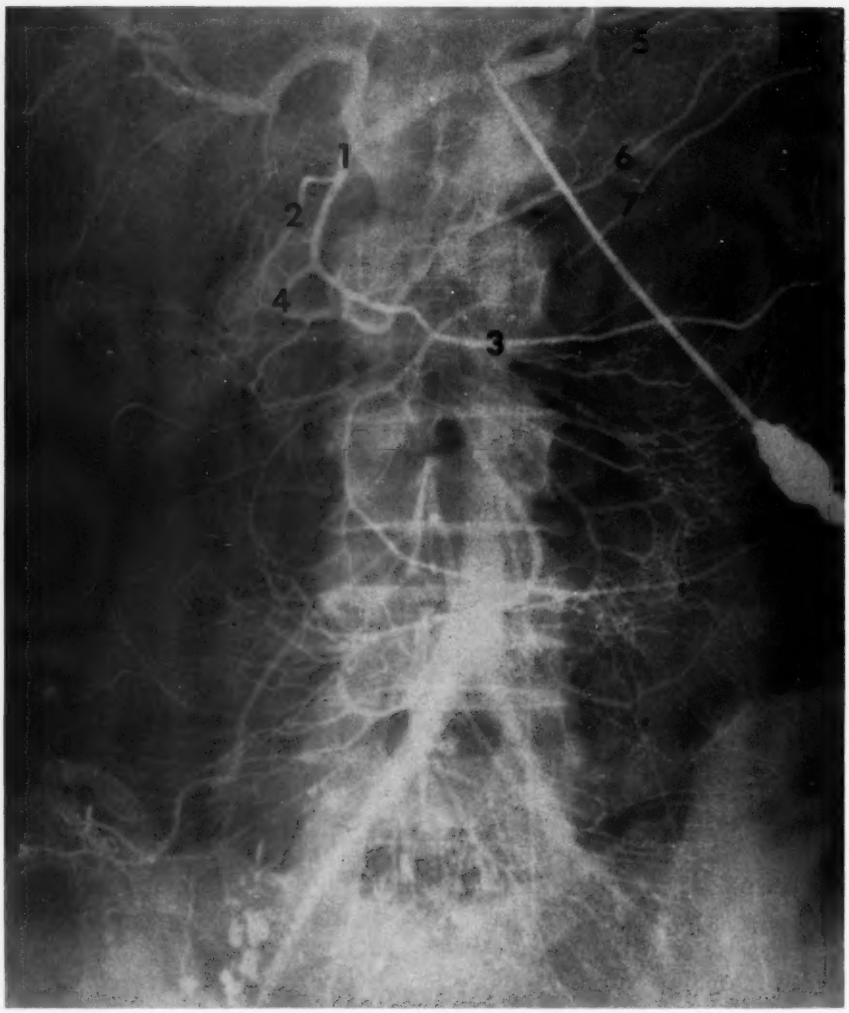

FIG. 4. Accidental selective injection of the celiac axis. The three major divisions of the celiac axis are easily identified. The trunk of the superior mesenteric artery is not opacified, although its terminal branches are well filled. The gastroduodenal artery (1) provides the posterior superior pancreaticoduodenal artery (2), and then bifurcates to form the right gastroepiploic artery (3) and the anterior superior pancreaticoduodenal artery (4). The pancreatica magna (5), branch of the splenic, and the inferior pancreatic artery (6) and the middle colic artery (7), branches of the superior mesenteric artery are well filled. The remaining arteries lying below the right gastroepiploic artery are intestinal branches of the superior mesenteric artery.

their curvilinear course and lack of branching. They occasionally arise from the renal arteries.

Segmental Branches. The segmental branches include the subcostal arteries and four pairs of lumbar arteries. The latter are most easily identified by their deep posterior divisions coursing through the paravertebral muscles to the back. Seen in axial view, these appear as a circular area of increased density immediately adjacent to their corresponding vertebral bodies.

ARTERIES OF THE PELVIS AND THIGH

The common iliac arteries arise as terminal branches of the abdominal aorta, usually opposite L-4, and, devoid of other branches, bifurcate to form the external iliac arteries and the hypogastric arteries. From the aorta at its bifurcation, a small middle sacral artery can sometimes be identified running inferiorly along the sacrum near the midline.

The external iliac artery, in its smooth curved course across the pelvis, gives rise,

immediately proximal to the inguinal ligament, to the inferior epigastric artery which courses superiorly and medially and to the deep circumflex iliac artery which courses superiorly and laterally.

The common femoral artery gives origin laterally to a small superficial circumflex iliac artery and medially to the superficial epigastric artery and to the deep and superficial external pudendal arteries. It is impossible to accurately identify members of this latter group, since the vessels are all of small caliber and are superimposed upon each other.

The common femoral artery terminates by dividing into the superficial femoral artery and the profunda femoris (Fig. 16). The former appears to be the direct continuation of the common femoral, descending in a long smooth curve in the medial portion of the thigh, providing several muscular branches and the highest genicular artery (anastomotica magna) in the proximal adductor canal. The profunda femoris courses laterally and inferiorly, providing near its origin the small medial circumflex and the larger lateral circumflex arteries. The latter vessel divides into a small superior branch which ascends lateral to the ilium and a larger inferior branch which descends to the knee along the lateral portion of the femur. A transverse branch supplying the region of the greater trochanter is sometimes identified. The main trunk of the profunda femoris descends laterally, giving off an inconstant number of perforating branches.

The branches of the hypogastric artery are extremely variable and unpredictable. Division of the hypogastric into an anterior and a posterior trunk is commonly observed anatomically, but difficult to identify roentgenographically. Initially, the posterior trunk provides one or two lateral sacral arteries which then subdivide to send segmental branches to each of the sacral foramina. Slightly more distally, the iliolumbar artery arises and courses superiorly, dividing into a lumbar and an iliac branch, the latter following closely the crest of the ilium. The remainder of the posterior trunk is continuous as the superior gluteal artery which passes laterally through the greater sciatic notch to supply the buttocks.

Fig. 5. Normal pelvic arteriogram.

The remaining arteries of the pelvis arise from the anterior trunk. The identification of these vessels is accomplished largely by tracing their courses proximally from their areas of distribution peripherally. The obturator artery can usually be identified by its terminal branches encircling the obturator foramen and by its pubic branches crossing the symphysis pubis. In 37 per cent of cases,[27] the obturator arises from the inferior epigastric artery, the deep circumflex iliac artery, or directly from the external iliac artery, and as such is known as an aberrant obturator. The internal pudendal artery characteristically crosses the obturator foramen below the obturator artery and may be identified by its terminal scrotal or labial branches. The inferior gluteal artery may arise in common with either the superior gluteal artery or the internal pudendal artery. It courses inferiorly and laterally and characteristically

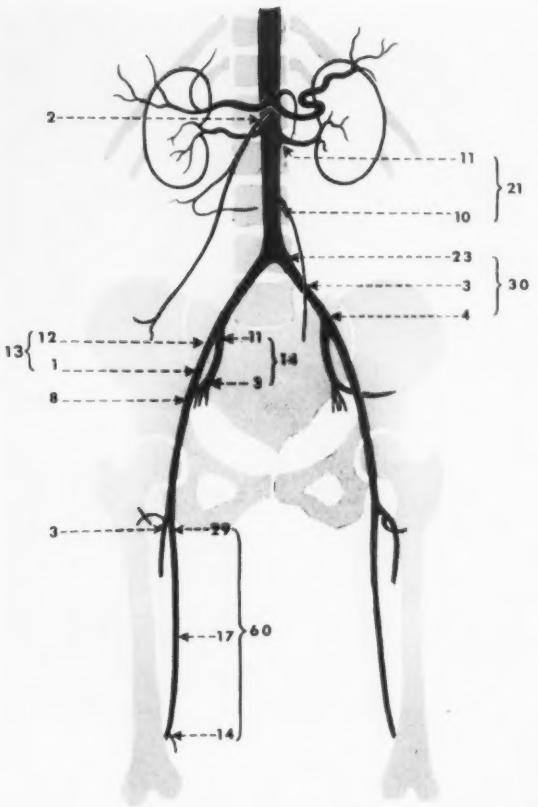

FIG. 6. The proximal site of obstruction in 151 arterial thromboses. The natural distribution of arterial obstruction is not represented since the entire arterial tree was not examined in every case.

runs superimposed over the femoral neck in much the same position as one sees metallic orthopedic nails used in the internal fixation of femoral neck fractures. The middle hemorrhoidal artery and the vesical arteries are small branches of the hypogastric artery, inconstant in their position and virtually impossible to identify accurately. The uterine artery, when present, may be identified arising high from the anterior trunk and terminating medially in a tortuous coiled network of vessels, the azygous arteries of the vagina and uterus. (See Borell and Fernstrom[6]).

PART II. COLLATERAL CIRCULATION IN OBSTRUCTIVE ARTERIAL DISEASE

The account of Leriche[19] in 1940 was the first of numerous reports describing thrombotic obliteration of the aortic bifurcation. Ever-increasing use of translumbar aortography[11,21,22,30,32] has demonstrated a previously unsuspected frequency of segmental occlusive disease in this and other major arteries. These circumstances have stimulated the development of a variety of methods for the treatment of obstructive arterial disease, namely, lumbar ganglionectomy,[14,25,26] aortectomy,[3,6,10] thromboendarterectomy,[4,31,35] arterial[7,16,17,28] and venous grafting,[15] and insertion of plastic prostheses.[36]

In the surgical treatment of obstructive arterial disease, it is desirable to know before operation (1) the level and extent of the arterial block, (2) the disease status of adjacent main-channel vessels, and (3) the adequacy and distribution of collateral circulation. Properly executed aortography can provide all this information.

As previously indicated, 151 arterial obstructions in 91 patients were available for

FIG. 7. Aortic obstruction just below the renal arteries (arrows). Principal collateral circulation arises in the middle colic artery (1) and passes to the left colic branch (2) of the inferior mesenteric artery (3) to enter the aorta below the block (4).

study. Thirty-one of these obstructions were incomplete, but all were severe, the majority having demonstrable collateral circulation. All the visible routes of collateral circulation around the major sites of obstruction were tabulated and will be described, but no reliable statistical analysis of the frequency of their development is possible because of the varying completeness of their filling. The distribution of the obstructions and their tendency to extend distally from the origin of major arterial branches is illustrated in Figure 6. Although not shown in this diagram, they characteristically terminate at the origin of a large vessel below, which is always an important part of the collateral circulation.

Aortic Obstruction. (19 complete; 2 incomplete). Of the complete aortic obstructions, 10 were immediately distal to the origin of the renal arteries, while 9 were at or distal to the origin of the inferior mesenteric artery. In all but 4 cases, the block extended at least to the bifurcation of the common iliac arteries; in a single case the lower level of the block was proximal to the origin of the inferior mesenteric artery.

When the level of block is above the inferior mesenteric artery, the predominant collateral pathway is the middle colic branch of the superior mesenteric artery leading to the left colic branch of the inferior mesenteric artery. From this artery, blood may return to the aorta if the origin of this vessel is patent (Figure 7), or, as is much more common, blood flows distally to the hemorrhoidal arteries and then to the hypogastric artery because the origin of the inferior mesenteric artery is above the lower level of the block. However, when the aortic block is distal to the origin of the inferior mesenteric artery, the middle colic artery is not involved, and blood is routed directly from the inferior mesenteric to the superior hemorrhoidal artery and its anastomoses with the middle and inferior hemorrhoidal arteries of the hypogastric system (Fig. 8). The vessels forming these channels are frequently dilated to two or three times their normal caliber, and, in most cases, appear to provide the principal collateral circulation. These vessels are well illustrated in Albanese *et al.*,[1] Price and Wagner,[30] and Lindstrom.[24]

Fig. 8. Aortic obstruction just below the inferior mesenteric artery (1). This artery and its superior hemorrhoidal branch (2) are grossly enlarged. The latter vessel provides extensive anastomoses in the perirectal tissues. Through one such anastomosis, retrograde flow through the middle hemorrhoidal artery (3) fills the left inferior gluteal artery (4).

Other significant collateral circulation arises in the segmental arteries, the intercostal (Fig. 9) and lumbar arteries (Fig. 10) in the posterior body wall. These anastomose with the deep circumflex iliac branch of the external iliac artery and with the iliolumbar and superior gluteal branches of the hypogastric artery. Although notching of the lower ribs has not yet been observed, it might be expected secondary to the enlargement and tortuosity of the intercostal arteries. On a single occasion, small intraluminal radiolucent defects were present at

FIG. 9. Aortic obstruction. The dilated, tortuous, intercostal arteries (arrows) are well filled. The hepatic artery and its branches and the right renal artery are easily identified. Intercostal collaterals are well illustrated by Elliott and Peck.

the expected point of origin of the inferior epigastric arteries (Fig. 10). We believe these are "mixing defects" representing the entrance of nonopacified blood arising in the internal mammary arteries and flowing retrograde through the inferior epigastric vessels to the external iliac arteries, much the same as it does in congenital coarctation of the aorta.

The adaptability of these segmental arteries is decisively shown in their striking reduction in size following thromboendarterectomy (Fig. 11).

Common Iliac Obstruction (14 complete; 16 incomplete). Obstructions of the common iliac arteries were isolated without evidence of occlusion above or below this level in 14 of 24 patients. In 6 of 24 patients, the obstruction was bilateral. The frequent observation of isolated blocks at this level and the general tendency of aortic obstruction to extend into these vessels supports previous reports[19,20] that aortic obstruction frequently begins in the common iliac arteries and extends superiorly. Of the 14 complete obstructions, 8 extended to the bifurcation of the common iliac, and the remaining 6 extended for an unknown distance, presumably further distally. (Unless collaterals fill the main stem arteries beyond the distal extent of the block, it is not possible to determine the exact length of the obstruction. This was true in many of our cases.) Of the incomplete obstructions, all but 2 were located either at the origin of the common iliac artery or at its bifurcation.

As with aortic obstruction, the principal collateral paths are the inferior mesenteric artery through its superior hemorrhoidal branch to the middle and inferior hemorrhoidal arteries of the hypogastric system and lumbar anastomoses with the iliolumbar artery, the superior gluteal artery, and the deep circumflex iliac artery. When the block is unilateral, significant collateral circulation arises between the various branches of the hypogastric and their fellows of the opposite side. Of these, the lateral sacral anastomoses (Fig. 12 and 14) and the obturator anastomoses (pubic branches) are the most significant (Fig. 14). Less frequently observed are those between the internal pudendal arteries (Fig. 13) and the middle hemorrhoidal arteries. In isolated cases, collateral paths were observed between the middle and lateral sacral arteries.

Hypogastric Obstruction (13 complete; 2 incomplete). Obstructions of the hypogastric artery invariably were associated with obstructive lesions elsewhere, principally in the common iliac and external iliac arteries. In only 2 of 12 patients was the obstruction bilateral. Since no examples of isolated hypogastric occlusion were encountered, the collateral circulation which was observed necessarily provided blood around two or more areas of obstruction, making evaluation of the specific collateral for the hypogastric block somewhat inaccurate. However, no new collateral paths were discovered, and most of the collaterals

Fig. 10. Terminal aortic obstruction. Extensive anastomoses are present between the third (1) and fourth (2) lumbar arteries and between these lumbar arteries and the iliolumbar (3) and superior gluteal arteries (4). The arrows indicate "mixing defects." See text.

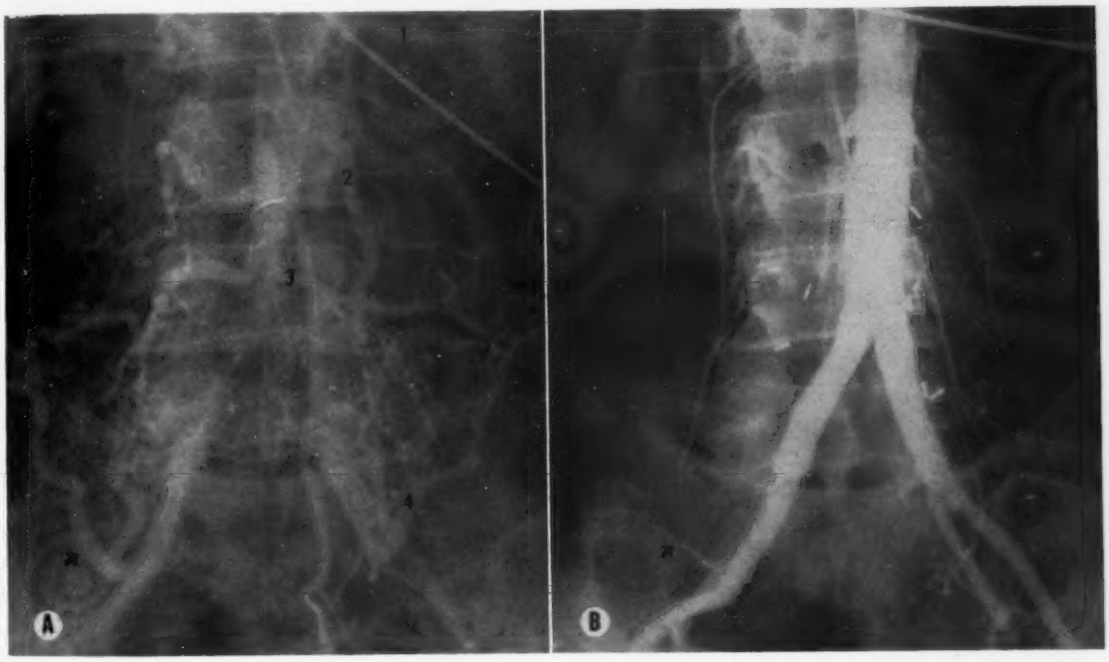

Fig. 11. Terminal aortic obstruction. (*A*) Preoperative aortogram demonstrating enlarged second lumbar arteries (1) and anastomoses between the third (2) and fourth (3) lumbar arteries and the iliolumbar arteries (4). (*B*) Aortogram four months following bilateral lumbar ganglionectomy and thromboendarterectomy demonstrating patency of the aorta and common iliac arteries. The regression in size of the second lumbar artery (upper arrow) and the iliolumbar artery (lower arrow) is remarkable.

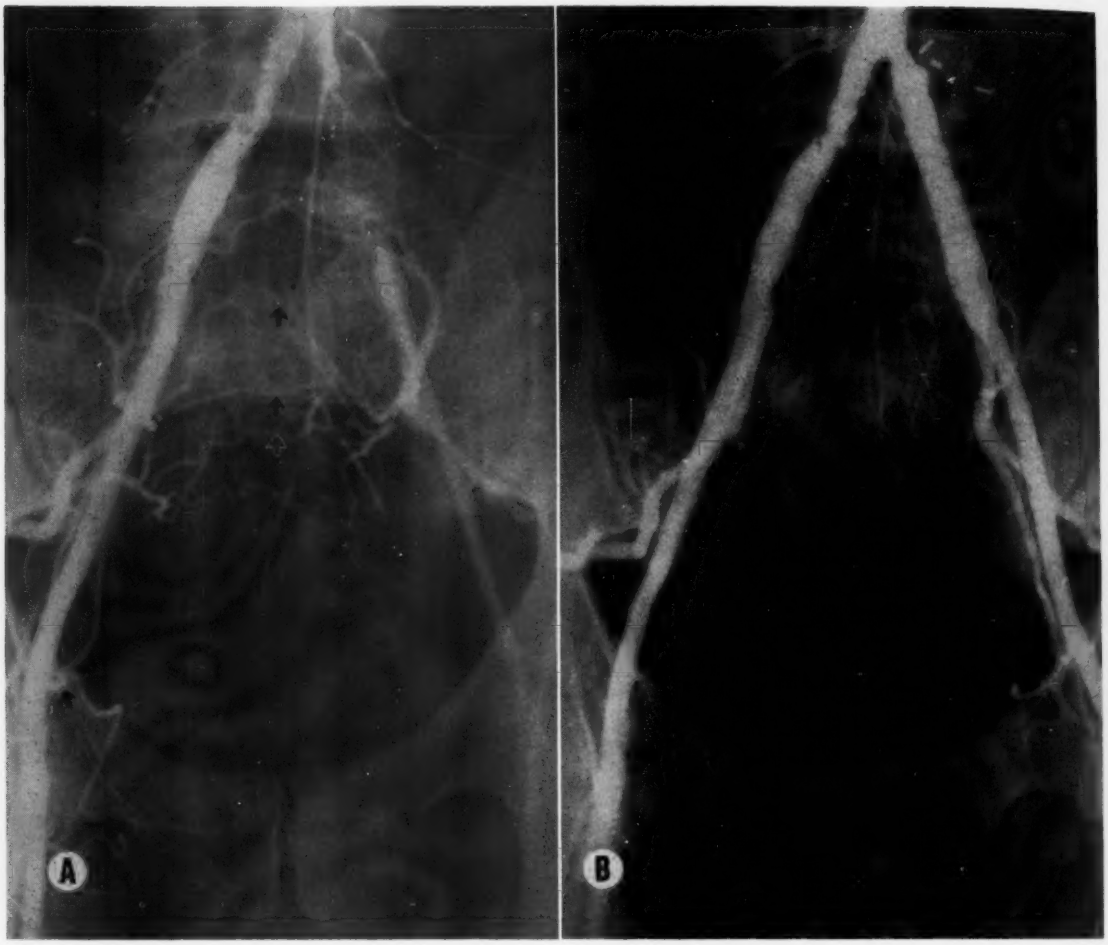

FIG. 12. Common iliac obstruction. (*A*) Preoperative aortogram demonstrating opposing lateral sacral anastomoses (central arrows). Notice the aberrent obturator artery (lower arrow). (*B*) Aortogram three months following left lumbar ganglionectomy and thromboendarterectomy. The common iliac artery is patent, and the lateral sacral anastomoses are no longer evident.

discussed and illustrated above under common iliac obstruction were represented.

External Iliac Obstruction (6 complete; 6 incomplete). As was the case with the hypogastric artery, obstruction of the external iliac artery was always associated with arterial block elsewhere. In all instances, the obstruction was unilateral and with one exception began at the origin of the external iliac artery.

The main collateral pathways are the superior gluteal to the lateral femoral circumflex artery and the obturator to the medial femoral circumflex artery. Of less importance are anastomoses between the inferior gluteal and the medial femoral circumflex artery and from the lumbar and iliolumbar arteries to the deep circumflex iliac artery.

Common Femoral Obstruction (5 complete; 3 incomplete). Obstruction of the common femoral artery was usually associated with obstruction at other levels, and was bilateral in 1 of 7 patients. However, the obstruction of the common femoral itself was frequently a short segmental block less than 5 cm. long.

Numerous collateral pathways are provided in common femoral obstruction. The most important of these is the inferior gluteal artery anastomosing with the profunda femoris (Fig. 15). In some cases, this

collateral vessel could be definitely traced to the medial femoral circumflex artery, but in others, the branch of the profunda femoris making the anastomosis could not be accurately named. Anastomoses between the obturator and the medial femoral circumflex artery are also frequently seen, while anastomoses between the superior gluteal and the lateral femoral circumflex artery are less frequently observed. Visible collateral sometimes exists between the terminal scrotal and labial branches of the internal pudendal artery and unidentifiable small collateral vessels in the medial portion of the thigh, possibly the external pudendal branches of the common femoral artery below the obstruction.

Fig. 13. Left common iliac obstruction. Anastomoses of opposing pubic branches of the obturator arteries (upper arrow) and opposing internal pudendal arteries (lower arrow) are well demonstrated. The position of the needle has resulted in fortuitously good opacification of the left internal spermatic artery (1).

Fig. 14. Incomplete left common iliac and complete left hypogastric obstruction. The terminal branches of the left hypogastric artery fill by anastomoses between the opposing lateral sacral arteries (upper arrow) and the opposing pubic branches of the obturator arteries (lower arrow).

Most anatomic texts mention the "crutiate anastomosis" in relation to blocks of the external iliac and common femoral arteries. A limited search did not disclose the source of this term nor uniform description of the vessels forming this anastomosis, although most authors emphasize the inferior gluteal and its connections with the lateral femoral circumflex artery and first perforating divisions of the profunda femoris.[2,9] We consider the cruciate anastomosis to be illustrated in Figure 15.

Superficial Femoral Obstruction (59 complete; 1 incomplete). Obstruction of the superficial femoral artery was bilateral in

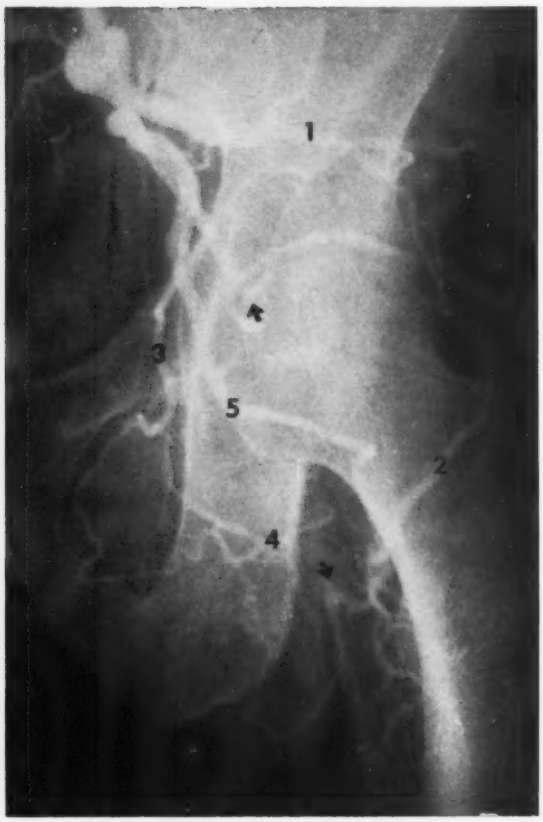

Fig. 15. Common femoral obstruction. The obstruction extends from the origin of the common femoral artery (upper arrow) into the distal superficial femoral artery (not shown), sparing the profunda femoris (lower arrow). Visible collateral is present between the superior gluteal artery (1) and the lateral femoral circumflex artery (2), between the obturator artery (3) and the medial femoral circumflex artery (4), and between the inferior gluteal artery (5) and the profunda femoris.

18 of 42 patients, and was associated with obstructive lesions elsewhere in 15 of the 42 patients. Of the 59 complete blocks, 28 occurred at the origin of the superficial femoral, 15 of these extending to the adductor canal and the remaining 13 extending for an unknown distance. Of the 17 complete obstructions originating in the mid-portion of the superficial femoral artery, 8 extended to the adductor canal while the remaining 9 extended for an unknown distance. The remaining 14 complete obstructions originated in the adductor canal, and their length was not ascertained. As may be appreciated from these figures, superficial femoral artery obstruction is comparatively lengthy, apparently because this vessel is devoid of large branches.

The collateral circulation in these cases is provided exclusively by the profunda femoris. At times the large bridging pathways (Krahl's "vital detours"[18]) can be identified as perforators, or the descending branch of the lateral femoral circumflex artery connecting with large muscular arteries near the adductor canal (Fig. 16). A frequently observed vessel of the latter type, the highest genicular artery (anastomotica magna), enters the main channel at about the level of the adductor canal. At other times, the profunda femoris supplies an extensive network of nameless

Fig. 16. Superficial femoral obstruction. The right superficial femoral artery is occluded from its origin to the adductor canal. The profunda femoris (1) provides numerous large muscular branches (2) which bridge the obstruction and join the superficial femoral artery in the adductor canal through the highest genicular artery (anastomotica magna) (3) and other nameless arteries below.

smaller vessels terminating in muscle. For further information, the reader is referred to Krahl's more extensive study of collateral circulation in this area.[18]

Deep Femoral Obstruction (2 complete; 1 incomplete). It has previously been reported that obstruction of the profunda femoris is rare.[23] The current series supports this observation. In each case the block of the profunda femoris was associated with obstructions elsewhere, and in 2 of the 3 patients, there was adjacent superficial femoral obstruction. No collateral circulation was observed in any of the cases due to inadequate arteriography.

Visceral Artery Obstruction (2 complete). Except for the following 2 patients, no convincing evidence of complete visceral arterial obstruction was observed. In these, the origin of the superior mesenteric artery was never visualized. In both, the proximal portion of the inferior mesenteric artery and its left colic branch were enlarged and supplied blood to the middle colic artery. The portion of the inferior mesenteric artery distal to the origin of the left colic artery was of normal size; aortic obstruction was not present in either case. We interpret these findings as indicating obstruction of the superior mesenteric artery proximal to the point of origin of the middle colic artery (Fig. 17). This may represent a congenital anomaly since in neither case is there historical evidence suggestive of previous vascular occlusion in the abdomen. Surgical or necropsy confirmation of this interpretation is lacking.

Fig. 17. Superior mesenteric artery obstruction. See text. Collateral circulation arising in the inferior mesenteric artery (1) passes to the superior mesenteric artery (not filled) through the left colic (2) and middle colic arteries (3).

DISCUSSION

The material in Part 1 on the normal roentgenographic anatomy of the arteries of the abdomen and pelvis is a composite of observations. Virtually every named artery of the abdomen and pelvis has been identified at some time. Because of incomplete filling and because of superimposition of vessels, the identification of certain arteries is not possible at all times. Anatomic variations have not been observed as frequently as they are reported in anatomy texts. However, most of these variations occur at the sites of origin of the main arteries, and it is in these areas that the roentgenographic identification is least accurate.

The current study of collateral circulation confirms previous anatomic descriptions of collateral channels[13,17,29] and does not disclose any new anastomoses.

The plethora of collateral pathways which are described above may be simplified somewhat. Around any arterial block, in the abdomen or pelvis, collaterals may form from one of three sources:

1. *Segmental and Parietal Arteries.* These include the intercostal, lumbar, inferior epigastric, and deep circumflex iliac arteries. Their anastomoses usually are with other segmental and parietal arteries on the homolateral side of the body, *e.g.*, intercostal to circumflex iliac, lumbar to lumbar, and lumbar to circumflex iliac.

2. *Visceral Arteries.* These include the superior and inferior mesenteric arteries

and their branches and the visceral branches of the hypogastric arteries. Through the parietal branches of the hypogastric artery, the obturator, superior, and inferior gluteal arteries, the visceral arteries may supply blood to the lower extremities. The mesenteric anastomoses form significant collateral paths in which the blood may flow antegrade or retrograde, depending upon the exact site of occlusion. This unique property makes these vessels highly important pathways in the presence of aortic and visceral arterial obstructions.

3. *Opposing Arteries.* These are the branches of the hypogastric artery, both visceral and parietal, which are capable of anastomoses with their fellows across the midline and are observed principally in unilateral pelvic artery obstruction.

The exact identification of all collateral vessels is usually not essential in planning the surgical treatment of arterial obstruction. However, at times, the life of small bowel, large bowel, and the extremities is maintained principally by a single visceral collateral (Fig. 7, 8 and 17), and the sacrifice of this vessel for any reason could prove disastrous.[24] With an accurate knowledge of specific collateral channels in such cases, the surgeon can more intelligently plan and execute operative procedures.

SUMMARY

A descriptive account of the roentgenographic anatomy of the arteries of the abdomen and pelvis as seen on translumbar aortograms is presented. Representative normal aortograms and diagrams illustrating the characteristic size, location, and distribution of the arteries of the abdomen and pelvis are described.

A study of the collateral circulation around obstructive arterial lesions of the abdominal aorta and its pelvic branches is reported and discussed. Illustrations of the major collateral pathways are described. The importance of accurate preoperative evaluation of collateral circulation is indicated.

CONCLUSION

The roentgenographic anatomy of the arteries of the abdomen, pelvis, and thigh as seen on 182 translumbar aortograms has been studied. The characteristic position, size, and distribution of the normal vessels, as well as some of the more commonly observed anatomic variations, are described. The collateral circulation occurring in about 151 arterial obstructions is described in detail. The importance of accurate preoperative evaluation of collateral circulation in certain instances is discussed.

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PERCUTANEOUS SPLENOPORTAL VENOGRAPHY*

UTILIZING RAPID SERIAL ROENTGENOGRAPHY

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IN THE surgical management of bleeding esophageal varices anastomoses between the portal venous and the caval venous systems have proved to be a most satisfactory treatment. By such a venovenous shunt the portal vein and its varicose tributaries in the esophagus are adequately decompressed.

Two types of anastomoses are available to the surgeon, the direct portacaval and the splenorenal shunt. The success of either procedure depends on the integrity of the portal circulation. Roentgen contrast visualization of this system makes it possible for the surgeon to choose his type of shunt and so to plan intelligently his incision and exposure.

The two methods of visualizing the portal system are: (1) portal venography developed by Blakemore and Lord[4] and later modified by Moore and Bridenbaugh,[10] and Child and associates;[6] and (2) percutaneous splenoportal venography first described experimentally in the dog by Abeatici and Campi.[1,2] The former method has the chief disadvantage of being restricted to the operating room. The examination is possible only after the incision has been made and the abdomen opened. This eliminates any flexibility in choice of incision. Furthermore, this technique does not usually provide complete coverage of the portal venous system. The latter method provides an excellent means of visualizing the portal system prior to operation. It is a simple, safe technique and prevents the possibility of many hours of fruitless search for a nonexistent or thrombosed vein. The most suitable vein is selected before the skin incision is made and it is directly and effectively approached. Despite these advantages, the method has received only limited clinical application.[3,5,7,8,9,11,12] The purpose of this paper is to describe our technique of examination and to add our own experience to the meager literature on the subject.

We have adopted the term splenoportal venography as best describing the roentgen character of this examination. The anatomy of the portal system has been excellently documented by Rousselot, Ruzicka and Doehner and is beyond the scope of this report.[11]

TECHNIQUE

After the patient is positioned on a wheeled stretcher in the regular anesthesia room in the operating suite, general anesthesia is induced. Endotracheal intubation is accomplished as this affords the anesthetist adequate control of the patient during the subsequent procedure. The patient is then transported to the radiologic department and positioned supine on a roll film magazine which utilizes 12 by 12 inch film (Fig. 1, *A* and *B*). The left arm is abducted 90 degrees and the left lower lateral chest is cleansed and prepared with aqueous zephiran. A No. 15 needle without an obturator is inserted in the midaxillary line in the ninth or tenth intercostal space, depending on the size of the spleen and the depth of the costophrenic sinus. As the needle is advanced, the patient is maintained in apnea to stabilize the diaphragm and to lessen the likelihood of lacerating the spleen. A sensation of firmness is imparted through the needle as it enters the spleen. It is then advanced 2–3 cm. farther into the substance of this organ in order to insure the introduction of the contrast medium well

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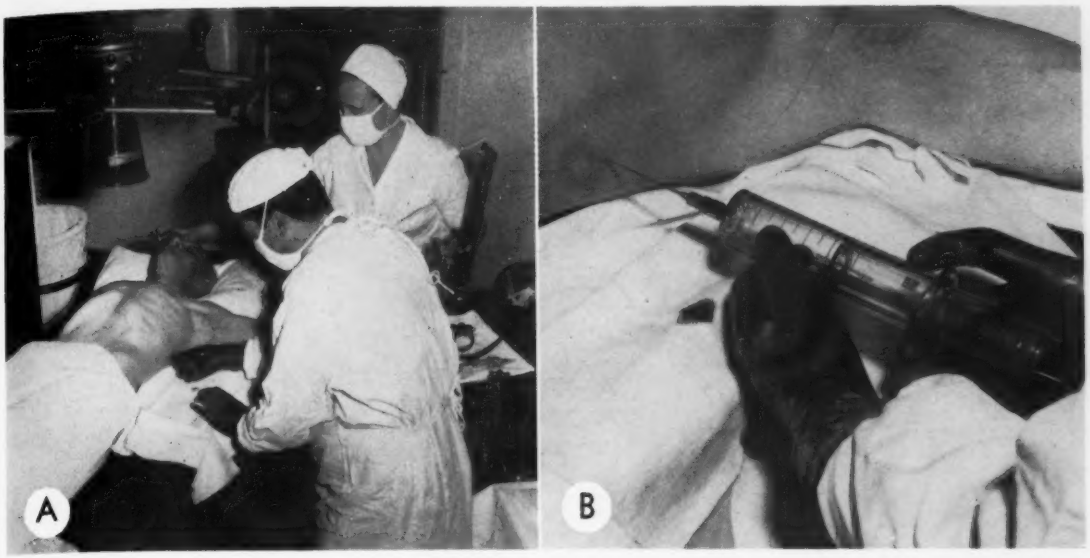


Fig. 1. (*A*) Patient positioned supine on roll film magazine. Needle has been properly inserted in spleen. Anesthesiologist maintains patient in apnea during insertion of needle. (*B*) Injection of contrast agent (urokon sodium 70 per cent 50 cc.). Injection done as rapidly as possible usually takes 5 seconds. Patient maintained in apnea until desired roentgenograms are secured.

into the pulp and to minimize subcapsular distribution. A free, slow flow of blood is further evidence that the needle tip is properly positioned in the splenic pulp. Shallow controlled respirations are permitted as a 50 cc. syringe filled with 70 per cent urokon is attached to the needle. The anesthetist is again asked to induce apnea, and, when this is achieved, the injection is begun and the roentgenographic exposures started. The forceful injection of the contrast agent requires about 5–6 seconds. On its completion the needle is withdrawn. Exposure rates of one per second for 12–15 seconds have provided complete coverage of the portal system. The roentgen factors are: 200 ma., 0.1 sec., 94 kv. peak for 20 cm. patient, distance 40 inches; the grid ratio of the magazine is 6.1.

Following completion of the examination the patient is returned to the operating room, the films are processed and read in a matter of minutes. If the puncture technique and injection of contrast medium have been satisfactory, the splenic and portal veins and major tributaries will be clearly demonstrated on the serial films.

RESULTS

Forty-four percutaneous splenoportal venographies were done on 41 patients. Three patients had the percutaneous puncture of the spleen and injection of contrast medium on the operating table, utilizing portable roentgen equipment and the single film technique. The remaining patients were examined in the radiology department on a 12 by 12 inch roll film magazine. Of the total number examined, 36 were cirrhotics with portal hypertension. Six patients had extrahepatic obstruction in the form of thrombosis of the portal or splenic vein (Fig. 2). One of the patients with extrahepatic obstruction, a seventy-three year old female with cirrhosis, developed a primary carcinoma of the liver which involved and thrombosed the portal vein (Fig. 3). There were 2 instances of splenomegaly secondary to blood dyscrasias. In each case examined definitive information regarding the status of the portal circulation was made available to the surgeon prior to operation (Fig. 4 and 5, *A*, *B*, *C* and *D*). In those patients who have ascites, ascitic fluid may drip from

FIG. 2. A forty year old Negro with portal hypertension, hepatosplenomegaly and esophageal varices. Admitted following massive hematemesis. A selected roentgenogram at 5 second exposure taken from serial examination. The portal venous system is outlined by contrast agent. There is uneven filling of the portal vein giving it a blotchy appearance. This is caused by a large thrombus in the vein which has not as yet caused complete obstruction.

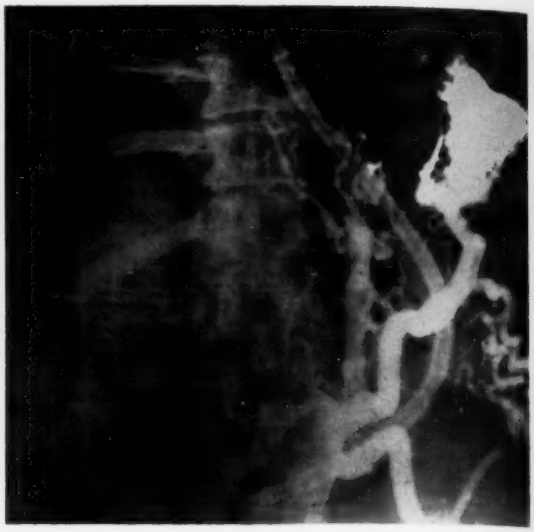


FIG. 3. Invasion and obstruction of the portal vein by primary carcinoma of the liver in a seventy-three old female with cirrhosis.

the needle before the spleen is entered and blood is obtained. The admixture of ascitic fluid and blood may well give an exaggerated impression as to the amount of blood loss incident to the puncture. Of the 26 patients who had a portacaval shunt, the spleens were left *in situ* and there has been no evidence of intraperitoneal bleeding in the postoperative period.

No immediate or delayed complications have occurred in any of the patients examined. In no instance has there been any change in the patient's vital signs during or subsequent to the injection. The pulse rate and level of blood pressure have remained stable. It is customary to find 50–100 cc. of blood in the left upper quadrant of the abdomen when the peritoneal cavity is opened. If the injection has been primarily subcapsular, the blood loss may be greater. The site of needle puncture may be visualized and in all but one instance there was no active bleeding at the time the

FIG. 4. A thirty-eight year old female with known biliary cirrhosis and esophageal varices. Admission precipitated by severe hematemesis. Selected roentgenogram at 6 seconds taken from serial examination. A Sengstaken-Blakemore balloon controls the hematemesis. Splenic and portal veins are of normal size, caliber and position. Arrow marks the site of entrance of superior mesenteric vein. At this point there is some loss in density of the contrast column caused by reflux of nonopacified blood from superior mesenteric vein.

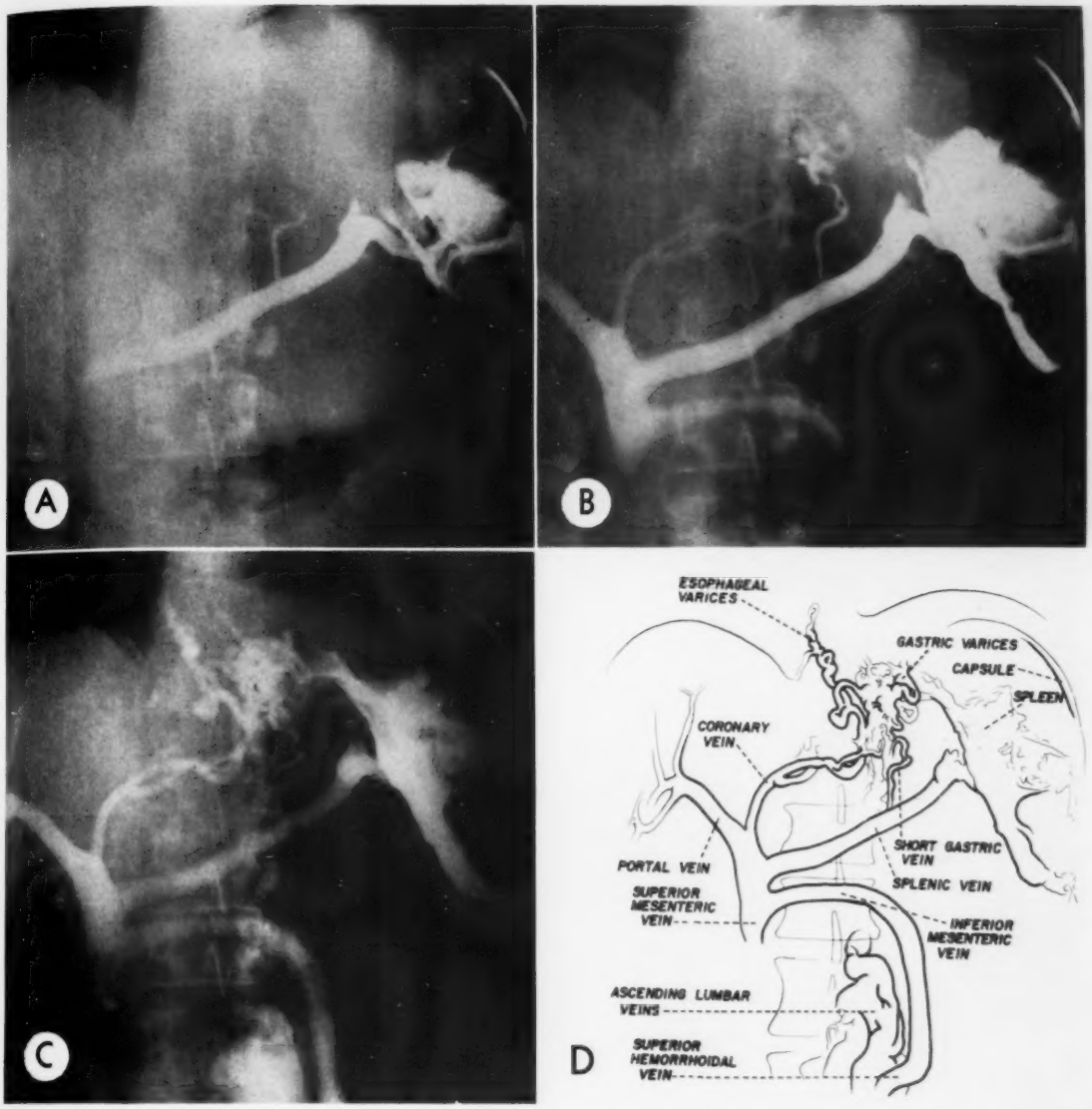


FIG. 5. (*A*) A fifty-three year old white female with Laënnec's cirrhosis, portal hypertension and esophageal varices. There was a history of several episodes of severe hematemesis. Roentgenogram at 3 seconds demonstrates early filling of the splenic vein. (*B*) Roentgenogram at 7 seconds demonstrates good filling of portal venous system and major tributaries. (*C*) Roentgenogram at 12 seconds demonstrates coronary vein, gastric and esophageal varices. The anastomoses between the inferior mesenteric vein and superior hemorrhoidal plexus is demonstrated. The latter is also seen to drain into the vertebral venous plexus (see tracing). (*D*) Tracing of *B* and *C*.

spleen was exposed. The one exception was a patient without portal hypertension who had an enlarged spleen and an obscure blood dyscrasia. The indicated treatment in this patient even without this additional factor was splenectomy, and this was done. The only other complication occurred during an attempt to visualize the patency of a previously performed portacaval shunt. On examination of the roentgenograms some of the contrast medium was found to be in the left pleural space. This resulted in pleuritic type pain and transient pleural reaction.

One of the more interesting variations in the relationship of the major venous

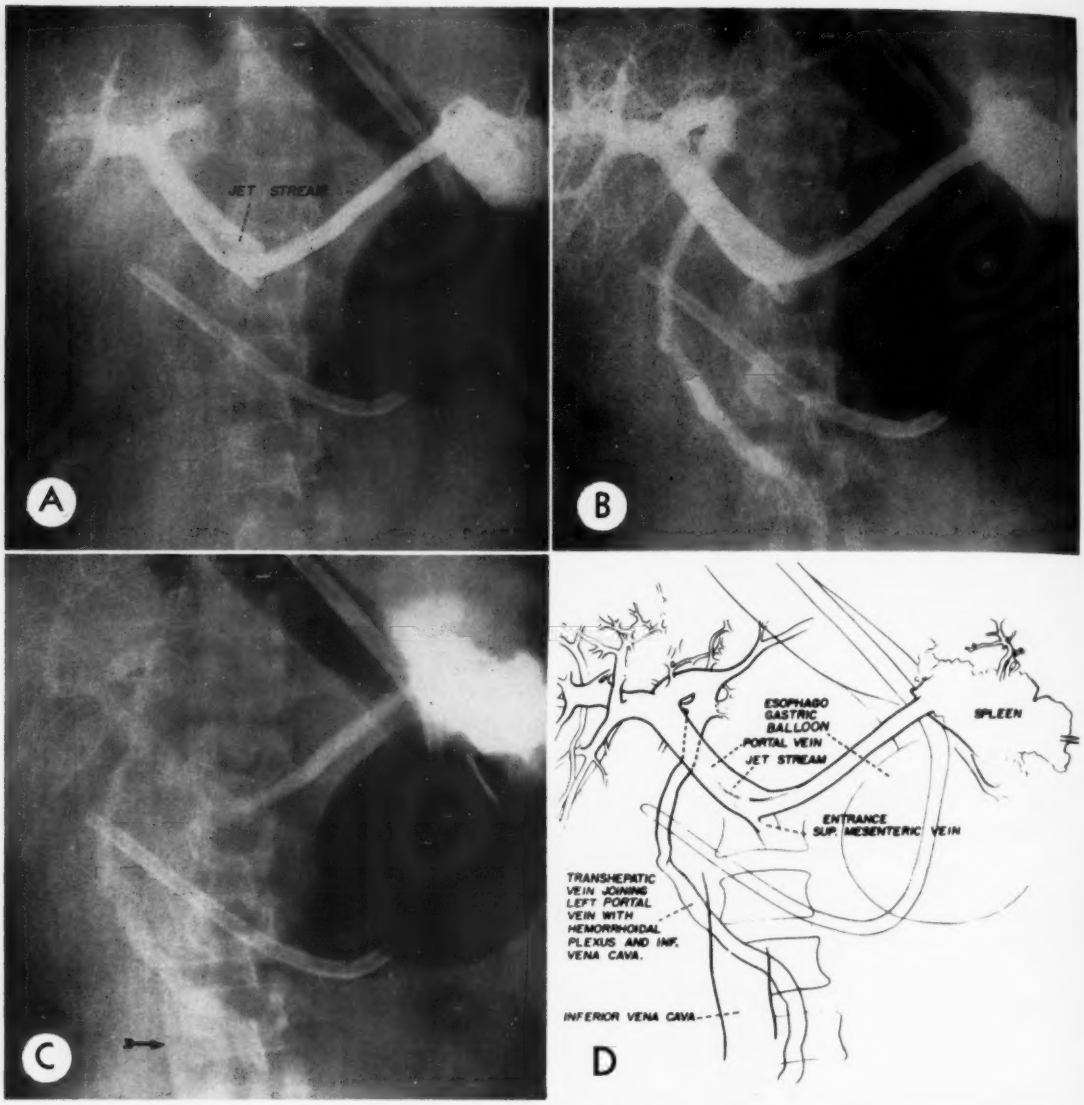


FIG. 6. (*A*) A seventeen year old girl with history of hepatitis and demonstrated esophageal varices. Selected roentgenogram at 3 seconds demonstrates a good injection into splenic pulp with rapid take-up of contrast medium. Splenic and portal veins are normal. The ribbon-like zone of decreased density marks entrance of superior mesenteric vein. (*B*) Further filling of the portal system at 7 seconds demonstrates a large transhepatic vein coming off the left hepatic branch of the portal vein and caudally anastomosing with the hemorrhoidal plexus. This in turn drains into the caval system as evidenced by opacification of the inferior vena cava. (*C*) Serial roentgenogram at 13 seconds shows opacification of the inferior vena cava (arrow). (*D*) Composite drawing of serial examination.

tributaries of the portal system is shown in Figure 6, *A*, *B*, *C and D*.

DISCUSSION

In the surgical management of portal hypertension, the method of percutaneous splenoportal venography provides invaluable preoperative information relative to the integrity of the portal circulation. In order, however, to avoid errors of interpretation and to insure complete coverage of the portal system, serial roentgenographic technique is essential. The 12 by 12 inch film roll covers the entire upper abdomen

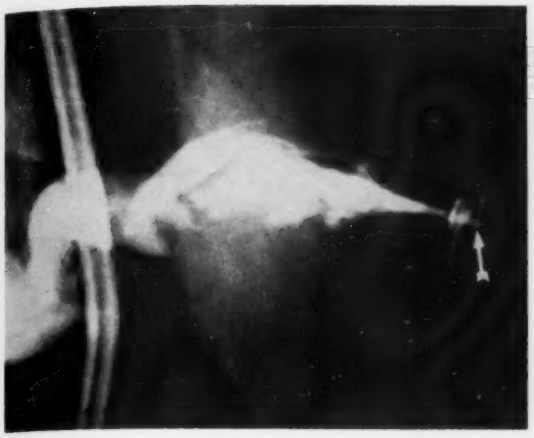


Fig. 7. Proper position of needle in spleen. It is well into the splenic pulp opposite the hilum. There is immediate take-up of contrast medium by splenic veins and no subcapsular reflux.

Fig. 9. A six year old girl with cirrhosis. Complete filling of intrahepatic portal veins.

even in very large patients. It covers the entire abdomen in children and small patients.

The diagnostic quality of the examination depends chiefly on the expertness of the splenic puncture. Optimally, the needle tip should be on a level with the hilum of the spleen and embedded about midway into the pulp (Fig. 7). Such a position will insure rapid take up of the urokon by the splenic veins and will prevent escape of any sizable amounts beneath the capsule. If the needle is inserted only a short distance into the spleen, the contrast medium during the injection will be forced back along the needle tract and collect beneath the capsule (Fig. 8). There appears to be little likelihood of exciting thrombophlebitis of the splenic veins as the contrast medium is first diluted by the splenic pulp.

Dreyer and Budtz-Olsen[8] in some of their cases noted a diminution of the opacified stream at the site of entry of the superior

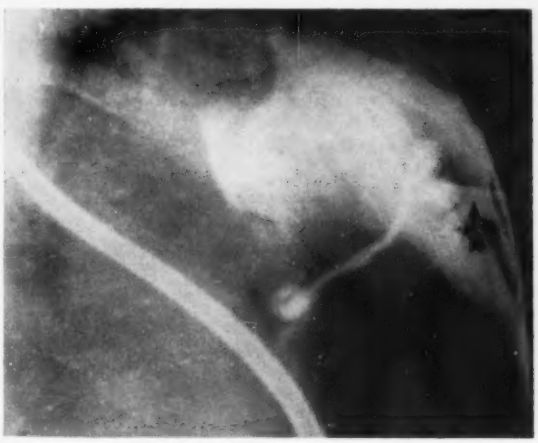


Fig. 8. Improper position of needle. It is too low and shallow, permitting backflow along the needle tract and subcapsular collection.

Fig. 10. A forty-two year old person with cirrhosis and hepatomegaly. Hepatic phase shows opacification of liver.

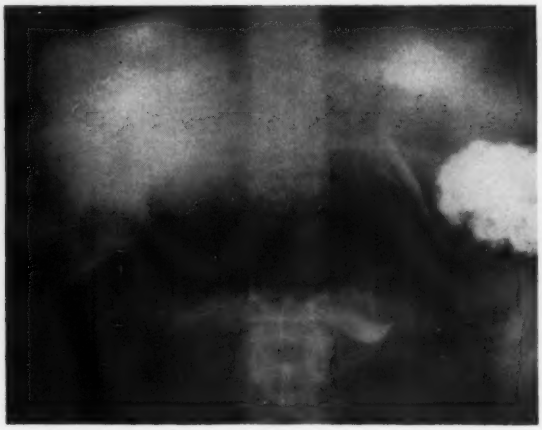
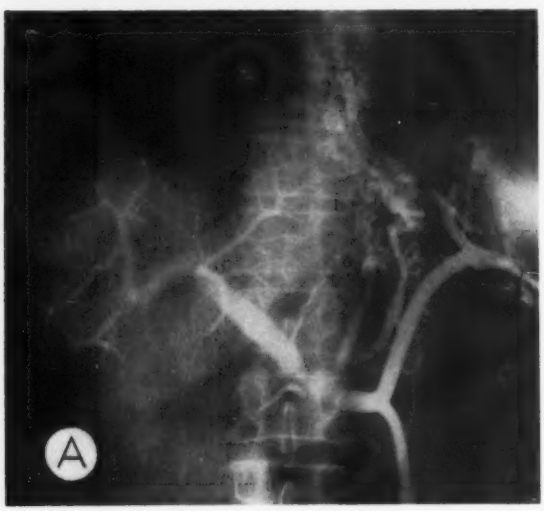


FIG. 11. A twenty year old female. Hepatic phase shows small shrunken, hobnail liver. The irregular blotchy filling defects in the opacified parenchyma represent the hobnail defects.

FIG. 13. A sixty-seven year old female with metastatic carcinoma of the liver. Hepatic phase demonstrates uneven and unequal opacification of inferior portion of right lobe of liver. The non-opacified areas corresponding to sites of metastases at exploration.

mesenteric vein. They felt this was due to the inflow of non-contrast blood from this vein. The decrease in density of the opacified column may create the impression of a filling defect. The phenomenon of the non-opacified jet stream at the site of entrance of the superior mesenteric vein is a common occurrence (Fig. 3, 4, 5 and 6*A*). This streaming effect, on occasion, may be so pronounced as to create the impression of a thrombus. Dilution of the opacified column at the junction of the superior mesenteric vein may be accentuated by a slow injection. The additional loss in density of the contrast column in the portal vein may simulate the irregular, uneven filling resulting from a partially thrombosed portal vein.

Gvozdanović and co-workers[9] state that in their experience the contrast agent left the spleen only as long as there was an elevated pressure in that part of the paren-



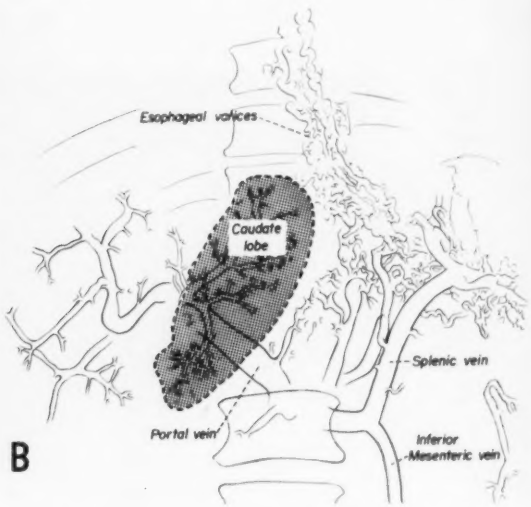


FIG. 12. (*A* and *B*) Spidery appearance of intrahepatic veins in patient with cirrhosis. Note opacification of caudate lobe of the liver.

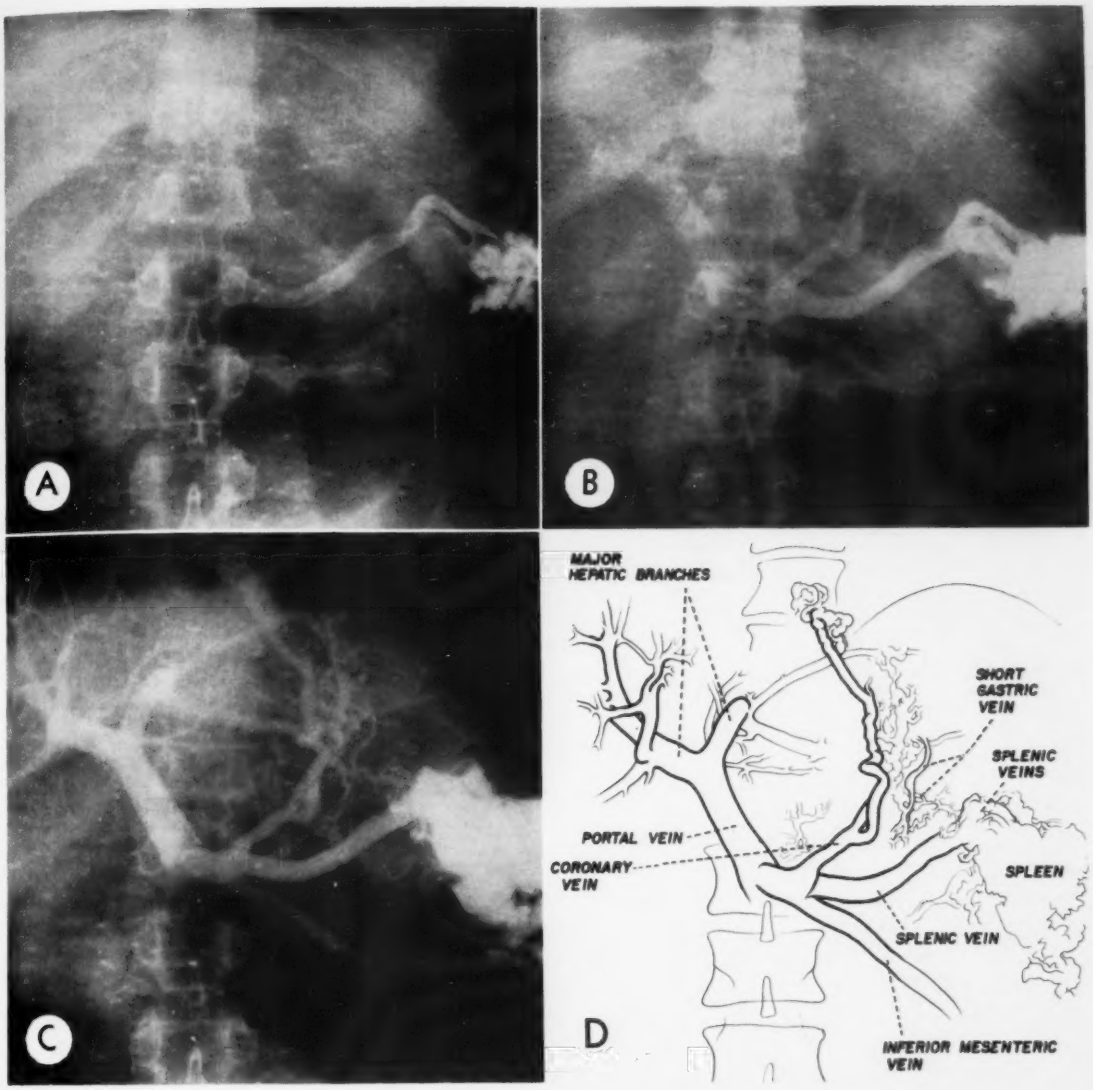


Fig. 14. (*A*) A fifty year old male cirrhotic with esophageal varices admitted because of hematemesis. Serial film taken at 4 seconds shows good position of needle in spleen and beginning opacification of splenic vein. (*B*) Five second sequence demonstrating complete filling of portal venous system. Note localized loss of contrast at site of entrance of superior mesenteric vein (arrow). (*C*) Roentgenogram at 10 seconds shows filling of esophageal varices. (*D*) Composite tracing of serial examination.

chyma into which the injection was being made. There is no doubt that the pressure of injection is responsible for the rapid take-up of contrast agent by the splenic veins and that the density of the circulating contrast medium drops off sharply on completion of the injection. It is our feeling, however, that the contrast agent continues to enter the portal circulation from the residue in the spleen. The fact that the contrast medium remains roentgenographically demonstrable in the portal system throughout the remainder of the examination seems to support this opinion. In the later phases of the examination, filling of the intrahepatic portal vein radicles and opacification of the liver parenchyma occur (Fig. 9, 10 and 11). This is another ad-

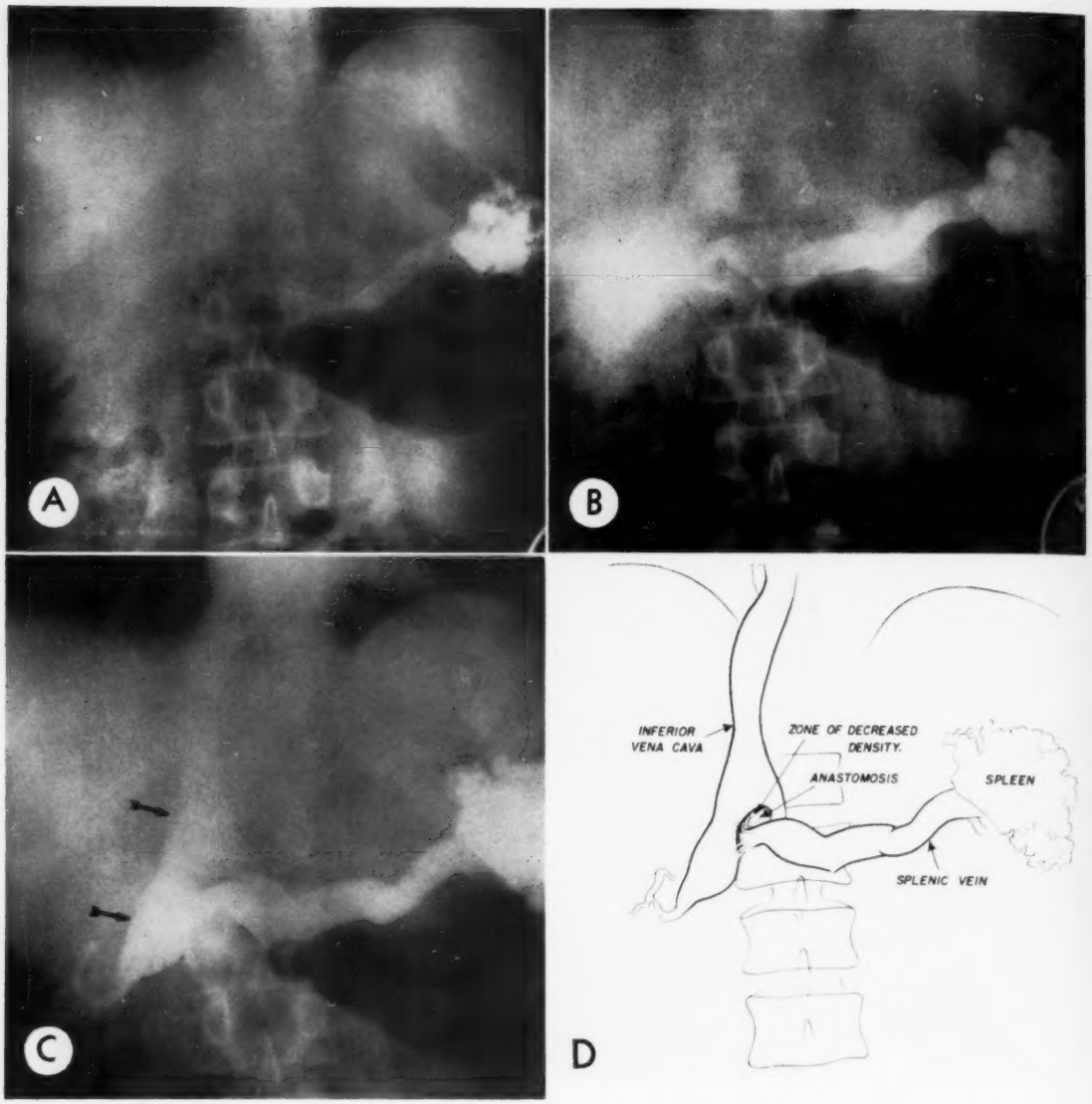


FIG. 15. (*A*) Postshunt examination. Good position of needle. Rapid take-up of contrast agent. Roentgenogram at 1 second demonstrates good filling of splenic vein. (*B*) Two second film demonstrates anastomosis. Note poor filling or lack of contrast at site of anastomosis. It is felt that this is due to eddies of noncontrast blood in vena cava at point of anastomosis. (*C*) Roentgenogram at 4 seconds shows inferior vena cava outlined by contrast agent with free and rapid flow of contrast blood from portal vein into inferior vena cava (arrows). The rapidity with which contrast medium disappeared was further evidence of the adequacy of the shunt. (*D*) Tracing of serial examination.

vantage of timed automatic serialization of films and may have practical significance in the diagnosis of liver disease. Mass lesions might be expected to show displacement of the intrahepatic portal branches. Disease resulting in destruction of liver substance may show as areas of nonfunctioning parenchyma. The intrahepatic portal veins in a number of patients with cirrhosis and hepatomegaly had a thin spidery appearance. In one patient the caudate lobe of the liver was distinctly visualized (Fig. 12, *A* and *B*). In another patient hepatic metastases were demonstrated in the right lobe of the liver (Fig. 13).

The technique was used in several des-

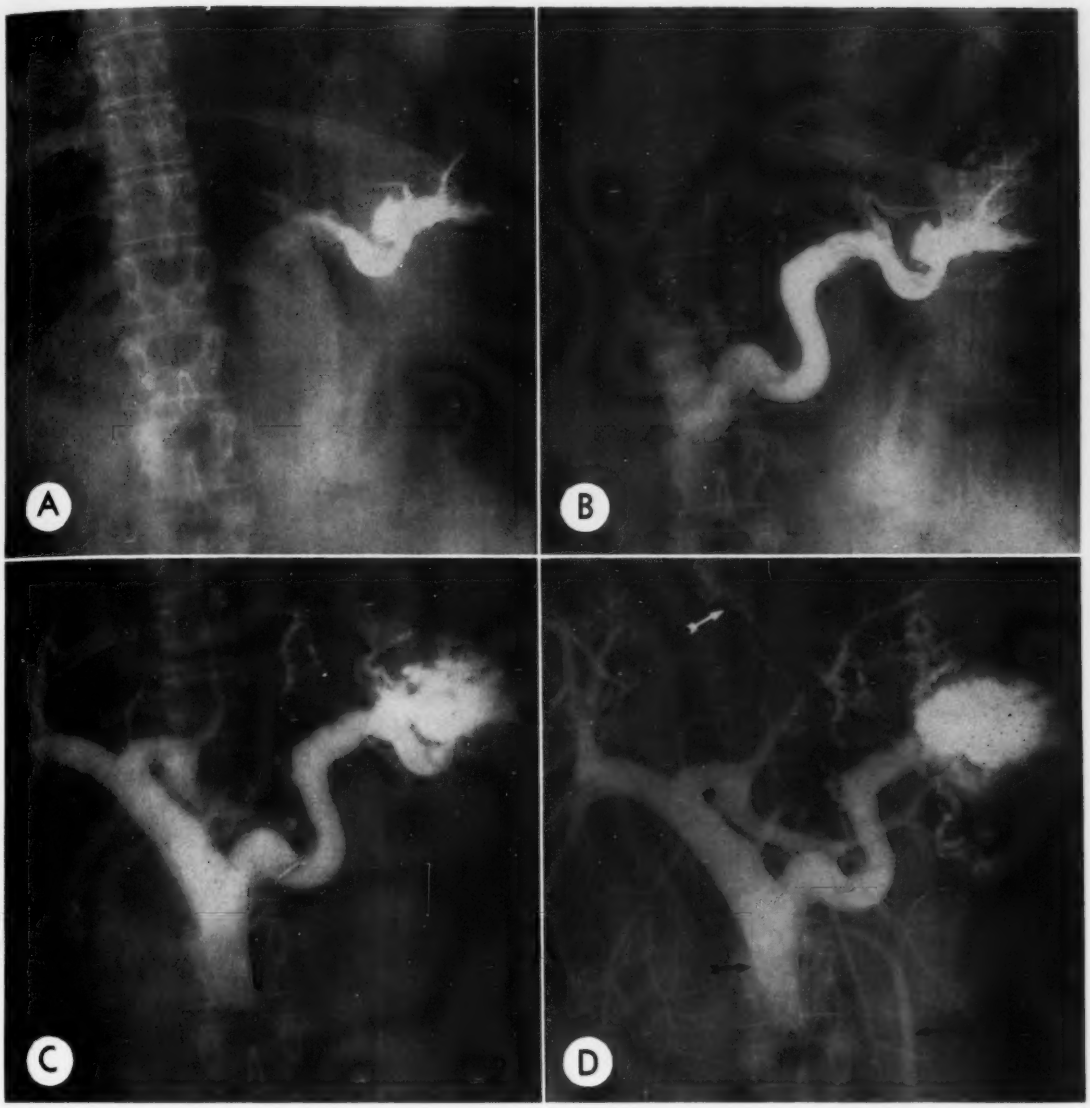


FIG. 16. (*A*) A forty-five year old female with complicated past history of several major abdominal operations including drainage of a pancreatic cyst. Roentgenogram at 2 seconds. Needle well positioned. Rapid take-up by splenic vein. (*B*) Work-up revealed patient to have cirrhosis, esophageal varices and hepatomegaly. There was a recent history of massive hematemesis. Roentgenogram at 4 seconds shows normal size, position and course of splenic and portal vein. (*C*) Roentgenogram at 6 seconds shows further filling of the portal system. (*D*) Roentgenogram at 10 seconds. Major tributaries of portal system filled. Varicosities about distal esophagus (upper arrow). The proximal stump of superior mesenteric vein visualized (lower left arrow). Inferior mesenteric vein somewhat smaller than usually seen (lower right arrow).

perately ill patients with Sengstaken-Blakemore esophagogastric balloons *in situ*. In each of these cases, though, there was good filling of the splenic and portal veins; the coronary, gastric and esophageal tributaries were not well demonstrated. This was contrary to the good filling of the major venous tributaries in the patients with portal hypertension who did not have balloons. It seems probable that despite a significant portal hypertension in each instance, the distended balloons prevented visualization of the varicose tributaries. An additional observation has been the failure

to visualize the superior mesenteric vein. In only one case was there enough reflux into the vein to identify its proximal segment. This lack of filling is a normal finding and should not be interpreted as an obstruction or block.

Cooper and associates[7] have used the percutaneous method of splenoportal venography to test the patency of a portacaval shunt. They have also applied the technique to the diagnosis of pancreatic and other retroperitoneal neoplasms which might displace, encroach on or block the portal or splenic veins. We have had the opportunity of using this technique to test the patency of a portacaval shunt in one case in our group of shunts.

REPORT OF CASES

CASE I. W. McG. No. 673 136. A fifty year old male with cirrhosis of the liver and esophageal varices was admitted to The New York Hospital for the first time on December 21, 1953 because of severe hematemesis and melena. A preoperative percutaneous splenoportal venogram demonstrated normal course, caliber and patency of the portal vein (Fig. 14, *A*, *B*, *C* and *D*). A portacaval shunt was done the same day. Preshunt portal pressure was 40.5 cm. H_2O. Postshunt portal pressure 14 cm. H_2O. He was discharged from the hospital after an uneventful postoperative course. Following discharge he was asymptomatic until March 27, 1954 when he had another episode of hematemesis for which he was re-admitted to the hospital. Roentgen examination of the gastrointestinal tract failed to identify a cause of bleeding. Esophagrams at this time failed to demonstrate the presence of varices. It was, nevertheless, thought that this shunt might have closed as a result of thrombosis. The percutaneous technique of splenoportal venography therefore afforded the opportunity of testing the patency of his shunt. This was done on April 13, 1954 and revealed a widely patent anastomosis between the portal vein and inferior vena cava (Fig. 15, *A*, *B*, *C* and *D*).

The intimate relation of the portal and splenic veins to the pancreas was of diagnostic importance in one of our cases.

CASE II. This was a forty-five year old woman with a complicated past history of several major abdominal operations including drainage of a pancreatic cyst. There was a recent history of massive hematemesis. Work-up revealed the patient to have cirrhosis, esophageal varices and a large mass, thought to be liver, filling the upper abdomen. The splenoportogram (Fig. 16, *A*, *B*, *C* and *D*) helped to eliminate the possibility of a pancreatic mass as there was no evidence of displacement or distortion of the splenic or portal veins.

Future experience with this new roentgen diagnostic technique may demonstrate that it will provide as much valuable information regarding pancreatic and other upper abdominal retroperitoneal neoplasms as it now does in candidates for surgical treatment of portal hypertension.

SUMMARY

The authors' experience with percutaneous splenoportal venography as a preoperative method of visualizing the portal venous system has been presented. The indications, advantages and technique have been reviewed. Representative cases have been illustrated and their interesting features discussed.

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ROENTGENOLOGIC FINDINGS IN SPLENIC PORTOGRAPHY

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IN PATIENTS who have an obstruction of blood flow in the portal venous system, the danger of the occurrence of massive and frequently fatal hemorrhage from esophagogastric varices is often present. Whipple found hematemesis as the first symptom in one third of early cases of portal block, and McIndoe found ruptured esophageal varices in 50 per cent of 26 cases of fatal cirrhosis. Those who were pioneering in shunt operations to relieve portal obstruction found remarkably little in the literature on the anatomy of the portal system. This segment of the problem was clarified by three studies that appeared in 1950: one in England[16] and two in the United States.[10,19] Valuable as these contributions necessarily were, a glance at anatomic representations of the portal venous system indicates that even a profound knowledge of the anatomic variations of this sytem may not be of much help to the surgeon who is trying to locate the site of a block in the portal or splenic vein. Neither will it assist him reliably, in the individual case, in a planned preoperative approach to the shunt procedure that may be lifesaving. And once the abdomen has been opened, he may find himself thwarted by a cavernomatous plexus of veins instead of a surgically useful portal vein.

What was needed was a method of opacifying the portal system so as to obtain a roentgenogram of the important anatomic features in the individual case, thus allowing a planned operative approach. Early attempts at injection of the portal veins after the abdomen had been opened were made by several workers,[18] but the establishment of operative portal venography was reported as a really practical procedure only in 1950 and 1951.[3,6,25,27,32] Portal portography is usually carried out by inserting a catheter into the superior mesenteric vein or one of its branches after laparotomy has been performed, injecting an opaque substance and taking a roentgenogram. This will give a good idea of the anatomy of the portal vein and, in portal hypertension or portal stasis, may show hepatofugal filling of the inferior mesenteric and the coronary or left gastric vein and of gastric and esophageal varices associated with the latter.

A more recent and simpler method for roentgenologic portrayal of the portal system is that of percutaneous splenic portography.[1,5,11,12] This procedure was accidentally performed in 1942, but the significance at the time was not stressed.[4] The efficacy of this method has now been confirmed by many authors.[2,8,14,15,18,20,21,24,28,30,34] In this procedure a needle is inserted percutaneously into the splenic substance, usually through the ninth intercostal space in the midaxillary line. Some workers prefer to perform it with the patient under general anesthesia, but most reports refer only to local anesthesia. The amount of material used varies from 10 to 50 ml. per injection. Opaque material is rapidly injected into the splenic substance. It passes immediately into the splenic vein and then into the portal vein

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and the liver. By this means the status of the main veins used in shunt procedures, namely the splenic and portal veins, can be readily judged.

To date, several hundred splenic portograms have been made. We know of no reported deaths, but in four instances serious complications have been reported. One was an instance of shock[7] and three were instances of splenic rupture requiring splenectomy.[13,34] Nevertheless, one is reminded that death may occur from splenic rupture following needle biopsy of the spleen.[20] A small amount of blood (25 to 50 ml.) may be found in the peritoneal cavity immediately after the procedure, but as often as not the site of puncture of the spleen cannot be discovered. A small area of necrosis occurs in the spleen, but after three weeks all that remains is a scar about 1 cm. in diameter.

Rousselot and co-workers[30] have recommended the routine use of both splenic and portal portography, but we have found that splenic portography may give sufficient information to the surgeon; in the event that it does not, portal portography can be done as an additional procedure.

Some authors have stressed the value of serial exposures[7,20,31] and, although we have as yet not been able to follow this suggestion, we believe it a useful feature, both in facilitating diagnosis and in avoiding pitfalls that are possible in the one-film technique. In a normal person the splenoportal circulation will carry all visible opaque material from the veins and through the liver in four or five seconds after the injection. Cooper and co-workers[7] have found that in portal block the opaque material may remain in the splenoportal system twenty-nine seconds and longer, this finding presumably being a reflection of portal stasis.

A corollary to this is the finding of Hunt[23] that the performance of a shunt operation in which the portal system is short-circuited to the systemic venous system will result in a great speed-up of venous flow out of the portal system. Thus, in a case of cirrhosis in which the speed of portal flow was 4 cm. per second before operation, it became 11.2 cm. per second after operation, which was even faster than the average normal flow of 9.4 cm. per sceond.

In this connection Hunt,[23] by measuring portal pressure manometrically and the rate of blood flow in the portal system by means of radioactive sodium and a double probe-pointed scintillation counter, has shown that portal stasis may occur in the presence of elevated or almost normal portal pressures and that in other cases there may be marked portal hypertension with no evidence of stasis of portal blood. Noting that the last-named situation was sometimes seen associated with the splenomegaly of Gaucher's disease or sarcoid, he postulated that this type of portal hypertension could be due to increased inflow of blood to the portal system as a result of enlargement of the spleen. In our experience some degree of portal hypertension has always been present when we have operated for bleeding thought to be from varices.

Cooper and co-workers[7] have demonstrated that in some cases of extrahepatic portal block, the opaque material tends to be cleared rapidly from the veins beyond the obstruction but to be retained longer in the veins distal to the obstruction (that is, closest to the site of injection). In this way, the actual site of an extrahepatic partial obstruction may at times be determined.

In connection with splenic portography, one is reminded of the finding of venous thrombosis in 80 per cent of patients in whom two injections of opaque material are made into a peripheral vein for angiocardiography.[9] The suggestion was made that one reason for the thrombosis might be injury to the vessel caused by raising the intravenous pressure rapidly to about 300 mm. of mercury. Splenic portography would seem to be safe from this point of view, as it has been found that the rapid injection of the opaque material into the spleen of the dog does not detectably increase the pressure in the portal vein.[11]

METHOD OF SPLENIC PORTOGRAPHY

To date we have performed splenic portography without portal portography in 42 cases without complications. In 2 cases the examination was unsuccessful owing to extrasplenic injection—no ill effects were noted. In another case visualization was unsatisfactory. We do splenic portography only in cases in which an operation is planned for the same day. In fact, the procedure is performed by the surgeon (Hallenbeck) after the patient is on the operating table under general anesthesia. The injection is made during a period of apnea or quiet breathing obtained by the use of curare or a similar drug. An 18-gauge needle attached to a 50-ml. syringe by way of a short piece of flexible plastic tubing (to decrease the chance of jarring the needle) is passed into the splenic pulp through the ninth intercostal space, in the midaxillary line. In the average adult, about 40 ml. of 70 per cent solution of acetrizoate (urokon) sodium is injected as rapidly as possible (four or five seconds), and a roentgenogram taken at the end of the injection. The film is developed immediately so that the surgeon can have information regarding the status of the vessels he will be using for the shunt procedure. We intend to change to a multiple-film technique as soon as we are able.

NORMAL SPLENIC PORTOGRAM

Rousselot and co-workers[30] have given a splendid description of the roentgen anatomy of the portal system. In the normal person, most of the opaque material leaves the spleen immediately and enters the splenic vein, flowing rapidly into the portal vein and through the liver (*hepatopetal* flow). The normal splenoportal circulation will carry most of the visible opaque material away in four or five seconds,[7] and there will be no evidence of reflux filling of such tributary vessels as the inferior mesenteric or left gastric veins. Only a small amount of opaque material lingers for a while in the splenic pulp. The junction of the superior mesenteric vein with the splenic vein may appear as an axial stream of decreased density in the opaque column in the portal vein, indicating the path of the incoming stream of blood.

ABNORMAL SPLENIC PORTOGRAM

The two main features of the splenic portogram that indicate abnormality are (1) reverse (hepatofugal) flow, that is, filling of tributary veins, especially the coronary (left gastric) and the inferior mesenteric veins, and (2) evidence of stasis.

Practically all patients with portal hypertension show evidence of backflow into the inferior mesenteric or left gastric veins or both. In our experience there is occasionally only the slightest evidence of hepatofugal filling on the roentgenogram, and it may be that if we had taken roentgenograms at other intervals of time following the injection, better visual evidence of hepatofugal filling may have been obtained. It is likely that in some patients with hepatofugal flow, there is actually a bi-directional flow in the portal system. Visual proof of this would seem to be the appearance, on the same roentgenogram, of hepatofugal filling of the coronary and inferior mesenteric veins, while at the same time there is evidence of blood flowing into the portal vein from the superior mesenteric vein (the radiolucent core seen in the opaque portal vein at the site of entry of the superior mesenteric vein).

However interesting such findings are, from the point of view of the surgeon who is to decide whether to make a right or a left thoracoabdominal incision (that is, whether to do a portacaval or splenorenal shunt) the anatomic appearance of the splenoportal venous system is the important feature. For this, splenic portography is an extremely valuable procedure. A few examples follow.

REPORT OF CASES

CASE 1. A sixty-four year old man had cirrhosis, congestive splenomegaly and bleeding varices. The splenic portogram (Fig. 1) indicated that the splenic and portal veins were

Fig. 1. Case I. Splenic portogram in a sixty-four year old man demonstrating widely patent splenic and portal veins. The surgeon was able to perform a splenorenal shunt. There is some hepatofugal filling of the coronary vein.

Fig. 2. Case II. Preoperatively, extrahepatic portal obstruction was suspected. The splenic portogram reveals both the splenic and the portal vein to be patent. Some of the opaque material is seen to have entered gastroesophageal varices. The surgeon was able to perform a splenorenal shunt.

patent, thus allowing the surgeon a choice of operation. There was retrograde filling of the left gastric vein, as well as opaque material in gastric varices.

Case II. Extrahepatic portal obstruction had been suspected preoperatively in a twenty-seven year old man. However, splenic portography (Fig. 2) disclosed a widely patent splenic-portal venous sytem, and reflux filling of the coronary vein and of gastric and esophageal varices. The surgeon was able to do a satisfactory splenorenal shunt.

Case III. A two and one-half year old boy had been suffering from bleeding esophageal and gastric varices and congestive splenomegaly. A preoperative diagnosis of extrahepatic portal obstruction was made, and it was noted that the child had suffered from neonatal omphalitis which had probably resulted in portal thrombophlebitis. The splenic portogram (Fig. 3) clearly showed a cavernomatous transformation of the portal vein, thus rendering it useless for a portacaval shunt. It was also obvious that the splenic vein was patent but small, but it was, however, the only vein that could possibly be used for a shunt procedure. At operation it was unfortunately found that the splenic vein was actually too small to be used for a splenorenal shunt. Splenectomy was performed, and it is not surprising that the child has bled again since the operation.

Case IV. In a fifty-four year old man the splenic portogram (Fig. 4) showed that the splenic vein was replaced by a plexus of veins and that it therefore was unsuitable for splenorenal shunt. A roentgenogram taken at a slightly later interval might have demonstrated the region of the portal vein more satisfactorily. Splenectomy was performed and the patient had no further bleeding.

Case V. A forty-six year old woman had cirrhosis of the liver, portal hypertension and bleeding esophageal varices. A splenic portogram (Fig. 5) demonstrated patent but small splenic and portal veins, retrograde filling of

Fig. 3. Case III. Splenic portogram in a two and one-half year old boy who had a history of neonatal omphalitis and bleeding esophageal and gastric varices. The splenic vein is patent but rather small. The portal vein is replaced by a plexus of veins. Gastric and esophageal varices are evident. There is subcapsular or capsular opacification of the spleen.

FIG. 4. Case IV. Preoperatively, extrahepatic portal block had been suspected and splenorenal shunt had been contemplated. The splenic portogram reveals that the splenic vein has been replaced by a plexus of veins unsuitable for a shunt operation. Splenectomy was performed.

the inferior mesenteric and left gastric veins, and esophageal varices. In the portogram the stream of blood from the superior mesenteric vein was represented in the portal vein by a radiolucent core. The film therefore suggested bi-directional flow in the portal system.

FIG. 5. Case V. Splenic portogram in a forty-six year old woman with cirrhosis of the liver, portal hypertension and bleeding esophageal varices. There is hepatofugal filling of the esophageal varices, the coronary vein and the inferior mesenteric vein. At the same time there is evidence of hepatofugal flow of superior mesenteric venous blood into the portal vein.

FIG. 6. Case VI. A thirty-four year old man with cirrhosis and hematemesis. The splenic and portal veins are large and patent. A portacaval shunt was performed. There is residual barium in the colon.

CASE VI. In a thirty-four year old man the portal pressure taken during operation was 520 mm. of water. The preoperative splenic portogram (Fig. 6) revealed little evidence of hepatofugal blood flow, and yet the preoperative roentgenogram after a swallow of barium showed marked esophageal varices. It is pos-

FIG. 7. Case VII. Cirrhosis of the liver, portal hypertension and bleeding esophageal varices in a forty-six year old man. The splenic and portal veins are patent and the large, serpentine shadow of the umbilical vein is seen. This was probably an example of the Cruveilhier-Baumgarten syndrome.

sible that if one or several more roentgenograms had been taken, hepatofugal flow might have been demonstrated. This coincides with our belief that more than one roentgenogram is required for satisfactory demonstration of the functional hemodynamic status of the portal system.

Case vii. A forty-six year old man was found to have esophageal varices and cirrhosis. Clinically, a well-marked caput medusae was manifested. A splenic portogram (Fig. 7) revealed widely patent splenic and portal veins; furthermore, a wide, tortuous umbilical vein could be identified. At operation the umbilical vein was found to be equal in size to the portal vein, and the pressure in the veins was found to be 275 mm. of water before a portacaval anastomosis was performed.

Unfortunately we did not listen for a venous hum over the abdomen. Nevertheless, it is felt that this case probably is an example of the Cruveilhier-Baumgarten syndrome (as distinguished from Cruveilhier-Baumgarten disease).[22]

Case viii. A five year old boy was brought to the Mayo Clinic with the diagnosis of bleeding duodenal ulcer and splenomegaly. Roentgenologic examination of the upper part of the gastrointestinal tract failed to show evidence of esophageal varices, but was thought to confirm the previous diagnosis of duodenal ulcer. Esophagoscopy disclosed small varices in the lower part of the esophagus. A splenic portogram (Fig. 8 *a*) revealed a splenic vein that bypassed the liver and apparently drained directly into the inferior vena cava near the site where the latter enters the right atrium—in other words, this patient had a natural splenocaval anastomosis. Adjacent to the site of the injection into the spleen, and in the region of the stomach, the roentgenogram showed a mass of opaque material. A laparotomy was then performed, and a portal portogram was made (Fig. 8 *b*). The portal vein entered the substance of the liver normally. However, the pressure in this vein was 400 mm. of water. Inspection and palpation of the duodenum showed no evidence of a duodenal ulcer. In view of the unusual anatomic features in this case, splenectomy alone was resorted to. Unfortunately and not unexpectedly, the patient subsequently has had further slight hemorrhage from the gastrointestinal tract. The desira-

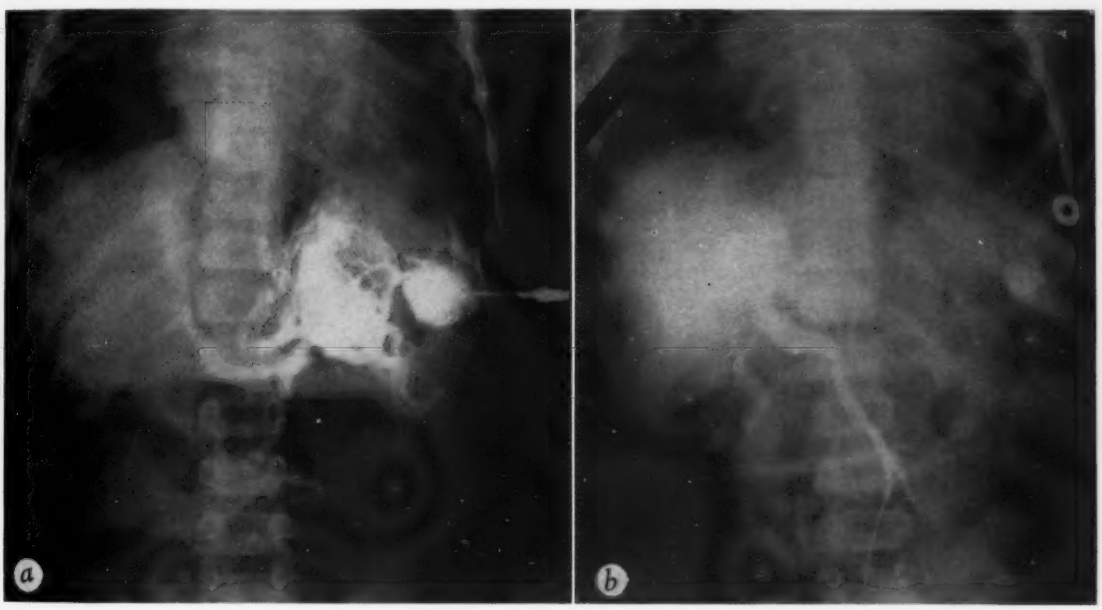

Fig. 8. Case viii. (*a*) Splenic portogram in a five year old boy with a history of duodenal ulcer and gastrointestinal bleeding. Note the peculiar plexus of vessels in the region of the stomach, and the fact that the splenic vein drains directly into the inferior vena cava in the region where it enters the right atrium. There is some retrograde filling of the inferior mesenteric vein, and there is probably some opaque material in the pulmonary vessels. This is a natural splenocaval shunt. See text. (*b*) Portal portogram of the same patient on the same day as in *a*. A normal connection between the portal vein and the liver is seen.

FIG. 9. Case IX. A sixty-two year old man who had partial obstruction of the portal vein by a thrombus. Note also the ease with which the opaque material enters the gastric and esophageal varices in spite of the Sengstaken-Blakemore tube.

bility of a portacaval shunt will have to be considered if hemorrhage becomes severe.

A roentgenogram of the thorax in Case VIII revealed anomalies of the second and third thoracic vertebrae. It has been shown conclusively that a relationship exists between cleft upper thoracic vertebrae and intestinal duplication,[17,33] and it is well known that patients with duplication may present with bleeding. But no evidence of such abnormality was found during the surgical exploration of the left upper quadrant. It is possible that the amorphous mass of opaque material seen on the portogram was in some anomalous retroperitoneal structure. Or it may be a reflection of cavernomatous transformation of a portion of the splenic vein.

CASE IX. A desperately ill sixty-two year old man was found to have tremendous esophageal varices as evidenced in a roentgenogram made after a swallow of barium. These varices had been the site of origin of massive bleeding. A splenic portogram was made (Fig. 9). The presence of a large thrombus that partially obstructed the portal vein was overlooked by us (it was subsequently found at operation). Additional interest was attached to this roentgenogram because it cast doubt on the efficacy of the Sengstaken-Blakemore tube that was in use at the time the splenic portogram was made. The opaque material was seen to flow unhindered into the gastric and esophageal varices.

Splenic portography has been of interest in indicating the extent of neoplasms of the pancreas and stomach,[18] and it is to be expected that additional cases will be reported.

Another method of incidentally outlining the portal venous system by the use of high abdominal aortography has been reported by Rigler and Olfelt.[29] This method may be of definite value in certain cases, although it may be less practical in the cirrhotic patient with ascites, who may find it extremely uncomfortable to lie prone. Its value would seem to be in patients in whom attempts are made to demonstrate masses in the liver, in patients with extrahepatic portal obstruction and in patients with small spleens.

SUMMARY

A short review of methods used to outline the portal venous system by opaque material is presented, and the procedure of percutaneous splenic portography is described in some detail. Nine cases are presented to demonstrate the practical aid that splenic portography may provide the surgeon.

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HEMANGIOMA OF THE LIVER DIAGNOSED BY SPLENOPORTOGRAPHY*

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AN IMPORTANT clinical procedure, splenoportography, which was introduced by Abeatici and Campi, proved valuable in diagnosing a case of hemangioma of the liver. Splenoportography is performed by using 15 cc. of 70 per cent urokon or diodrast injected percutaneously into the splenic parenchyma, employing a 17 gauge needle. The injection is performed under local anesthesia, using the ninth or tenth intercostal space in the midaxillary line. The needle is directed upward, medially and posteriorly. The radiopaque solution is injected rapidly (2–3 seconds) and serialographic roentgenograms are secured, usually at least six films in a period of 12 seconds following the injection. This yields a visual portrayal of the hemodynamics of the entire portal circulation and may demonstrate either congenital anomalies or thrombotic obstructive lesions. A number of other pathologic lesions may manifest themselves. Both cavernous and capillary hemangiomas are found in the liver, and may be single or multiple. These are usually not discovered during life but are revealed at autopsy. Brown and Gaspari excised pedunculated hepatic angiomata from adult patients. One of these cases was mistaken clinically for a floating kidney.

Splenoportography is gaining increasing importance as an aid in the differentiation of intra- and extrahepatic portal block in the production of portal hypertension. Portal venous stasis may result in a protean clinical picture ranging from nondescript meteorism to digestive hemorrhage with splenomegaly and hypersplenism. It is, therefore, of utmost importance, to define graphically the site of portal block in order to decide upon the proper form of surgical therapy through this procedure (Fig. 1, 2 and 3).

Portal hypertension may be caused by thrombosis of the portal vein or by intrahepatic obstruction such as cirrhosis of the liver. Symptoms may manifest themselves as gastrointestinal hemorrhage, hypersplenism or hepatic insufficiency. Infectious hepatitis or toxic agents may also be productive of an intrahepatic portal hypertension. Thrombophlebitis of extrahepatic veins may follow regional infection. According to Gross, omphalitis in infancy may result in portal vein obstruction secondary to infection traveling along the umbilical vein to invade the portal vein. Among other causes of extrahepatic block may be congenital venous valves in radicles of the portal vein.

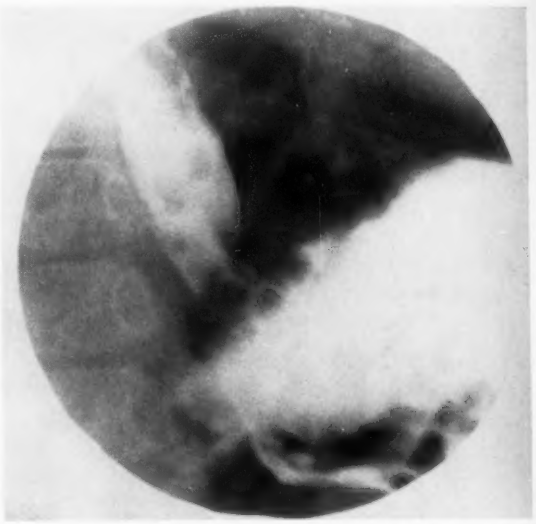

FIG. 1. Note large esophageal varices in a two and one-half year old child.

The case illustrated on the roentgenograms is that of a two and one-half year old female child who has had one attack of hematemesis. An esophagram showed the presence of varices. There was pronounced

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Fig. 2. Splenoportogram—four seconds after initiating injection—splenic and portal veins well outlined. Beginning visualization of hemangioma.

splenomegaly associated with hypersplenism. At operation a hemangioma in the porta hepatis was found. There was also marked phlebectasia in the gastroesophageal veins and in the veins of the calciform

Fig. 3. Complete visualization of cavernous angioma in the porta hepatis with complete delineation of intra- and extrahepatic portal circulation.

Fig. 4. Some diodrast puddling in the cavernous angioma; note clear outline of inferior vena cava.

ligament. A ligation of the splenic artery was performed to reduce the amount of portal blood flow. If clinically unimproved, a splenorenal shunt is contemplated when the child is four to five years old to yield greater likelihood of patency of the shunt (Fig. 4).

CONCLUSION

A hemangioma of the liver in a child was diagnosed by means of splenoportography. The case was complicated by portal hypertension and hypersplenism. The cavernous hemangioma of the porta hepatis may represent a congenital anomaly or a recanalization of an old thrombus.

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AN ANGIOGRAPHIC CASSETTE-CHANGING DEVICE*

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AN INEXPENSIVE and simple angiographic cassette-changing device, which is free of many of the shortcomings of other similar instruments, has been in use in our hospitals during the past three years.

The instrument is capable of handling either 14×17 or 10×12-inch cassettes of the ordinary type, and it could be adapted

FIG. 1. Part *A* of the platform mounted on the wheeled cabinet support. The hoppers are shown with the removable 10×12-inch hopper inserted into the permanently mounted 14×17-inch hopper.

to any other cassette size if desired. The moving Potter-Bucky grid is utilized, and therefore roentgenograms of the highest possible quality are obtained. The device operates on a simple mechanical principle, and breakdowns rarely occur. Films can be exposed as rapidly as one per second or at any slower rate desired, and up to twelve films can be taken during a single angiographic procedure. The instrument is adaptable to almost any roentgen-ray table which has a reciprocating grid and which has the Bucky tunnel open at *both* ends, *i.e.*, on both sides of the table.

CONSTRUCTION

The device consists of several parts:

1. A platform, consisting of two sections (parts *A* and *B*), designed to extend through the Bucky tunnel (Fig. 1, 2, 3, 6 and 7).
2. Hoppers to hold the unexposed cassettes (Fig. 1, 3 and 4).

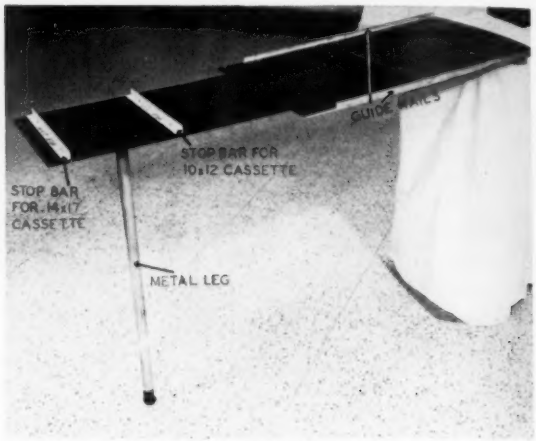

FIG. 2. Part *B* of the platform. The right hand end of Part B shown here is screwed to the left hand end of Part *A* (shown in Figure 1) when the unit is assembled for use. Note the position of the stop-bars (the one at the end is mounted permanently and the other is removable).

3. Plungers for propelling the cassettes (Fig. 3, 5 and 7).
4. Stop-bars for arresting motion of the cassettes (Fig. 2 and 3).
5. A micro-switch to activate the exposure at the proper times (Fig. 1, 5 and 7).
6. Protective lead shields (Fig. 7).

The measurements given in the diagrams are those of the machines used at our hospitals. Some variations of these dimensions

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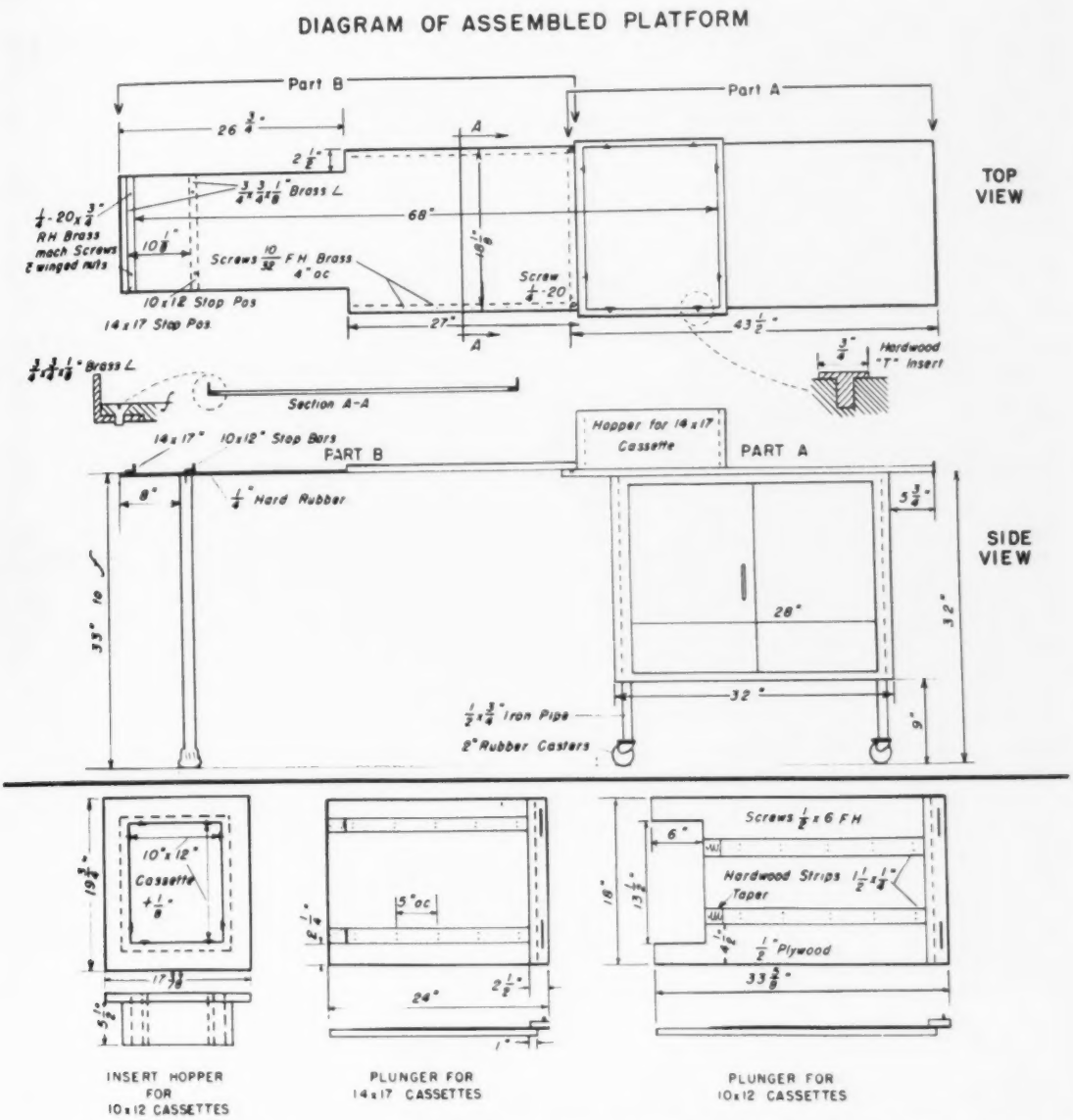

Fig. 3. Diagrams of the entire platform (Parts *A* and *B* assembled), of the insert hopper for 10×12-inch cassettes, and of the plungers.

may be necessary in order to adapt the apparatus to other roentgen-ray tables.

In describing the components of this equipment, reference will frequently be made to the functions of the various parts. Some of these points may not be readily understood in the description of the various components, but should be clarified in the discussion of the assembly and operation later.

The *platform* provides a smooth sliding

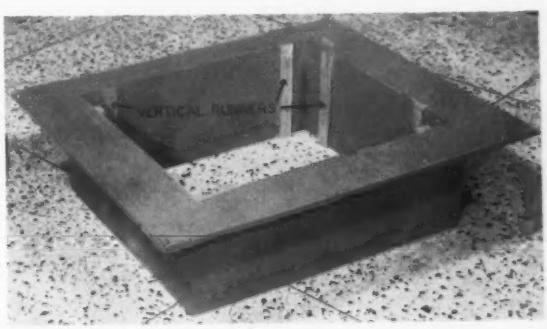

Fig. 4. Photograph of the insert hopper.

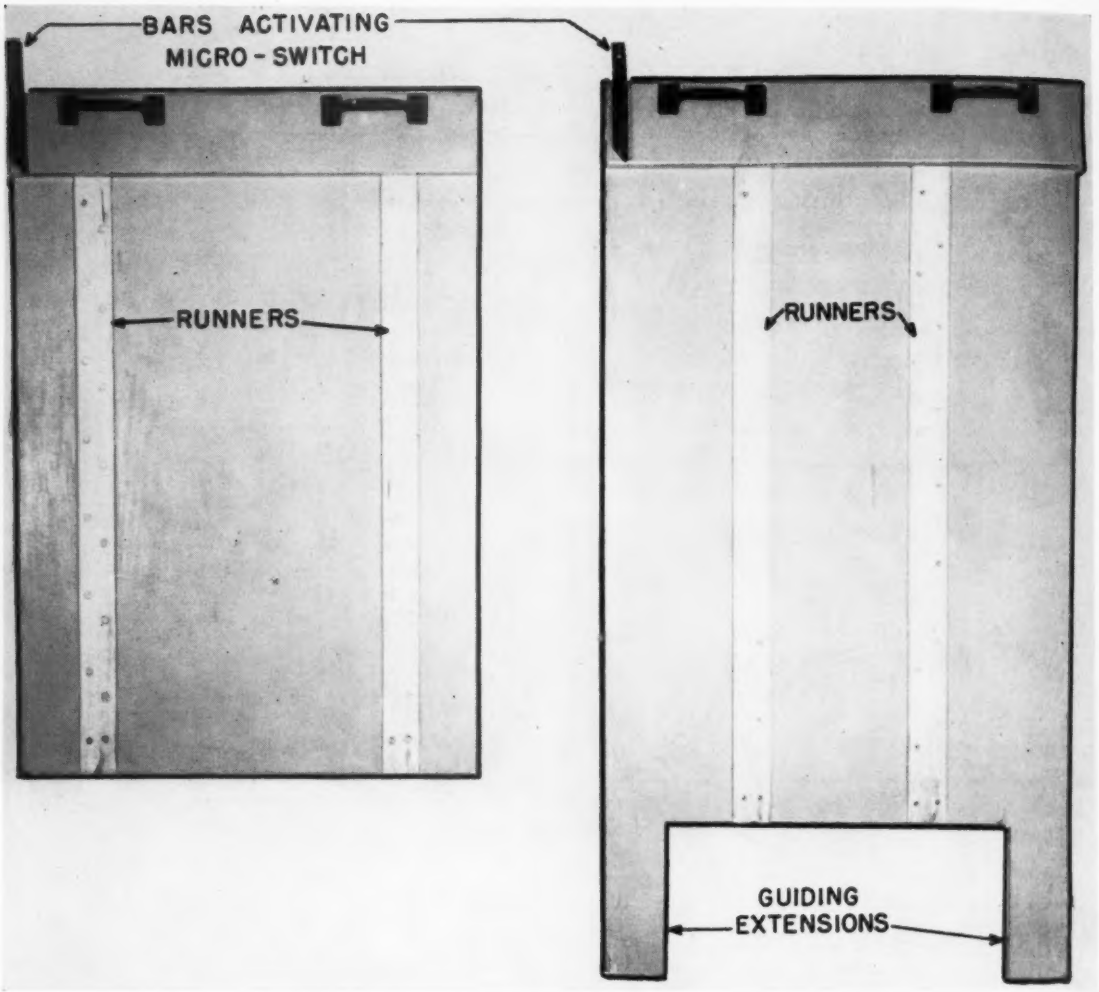

Fig. 5. Photograph of the plungers.

surface for the plungers and cassettes. It is made of a sturdy, hard, smooth material, such as Formica, and is about one-fourth of an inch in thickness. The platform is divided into two sections in order to facilitate the setting up of the apparatus. One part (part *A*, Fig. 1 and 3) is permanently mounted on a wheeled cabinet support (which also serves as a storage place); the second part (part *B*, Fig. 2 and 3) is designed to extend through and beyond the Bucky tunnel and is supported at its end by a removable leg (Fig. 2 and 3). When the unit is assembled, the two sections of the platform are joined together by means of two screws, thus providing a continuous smooth surface. Elevated brass rails (Fig. 1, 2 and 3) extend along both sides of the platform and are designed to keep 14×17-inch cassettes and the plungers properly aligned. These guide rails do not extend quite to the end of the platform, so that easy removal of the end cassette will be possible. For the same reason, that portion of the platform extending beyond the rails is somewhat narrower than the rest of the platform.

The *hoppers* hold the pile of unexposed cassettes. The hopper designed for 14×17-inch cassettes is permanently mounted to the cabinet across part *A* of the platform. Between the bottom of the walls of the

hopper and the surface of the platform are openings with enough clearance to permit the passage of one cassette or of a plunger (which has about the same thickness as a cassette). The wall of the hopper facing the roentgen-ray table is lined with lead 2 mm. thick, in order to protect the unexposed cassettes from direct and scattered radiation. The inner surface of the hopper (and also of the insert hopper) is provided with vertical runners (Fig. 3 and 4) which help to eliminate friction between the walls of the hopper and the dropping pile of cassettes. The height of the hopper is such that 10 or 12 cassettes may be stacked in it.

The removable *insert hopper* is used to adapt the machine for 10×12-inch cassettes (Fig. 3 and 4). This hopper is fitted into the larger one and rests upon it by means of overhanging extensions. The clearance space between the bottom of the walls of the insert hopper and the surface of the platform must also be slightly in excess of the thickness of one cassette.

The wooden *plungers* are used to push the cassettes. There are two plungers, one for 14×17-inch cassettes and the other for 10×12-inch cassettes. Each plunger is of such a length that, after complete insertion, it will advance the lowermost cassette from the bottom of the pile in the hopper to a position exactly underneath the center of the roentgen-ray table. An elevated board is attached to the main part of each plunger, and serves to act as a stop for the plunger at its maximum inward position. The plunger handles are mounted on this board. When completely withdrawn, the plungers clear the hopper and permit the pile of remaining cassettes in the hopper to drop upon the platform. The plunger for 10×12-inch cassettes is equipped with two guide extensions whose purpose is to steer the cassettes down the middle of the platform (Fig. 3 and 5). Each plunger is equipped on its surface with two elevated runners which are tapered at the end contacting the cassettes (Fig. 3). The purpose of these runners is to reduce friction between the plunger and the pile of cassettes remaining above it in the hopper.

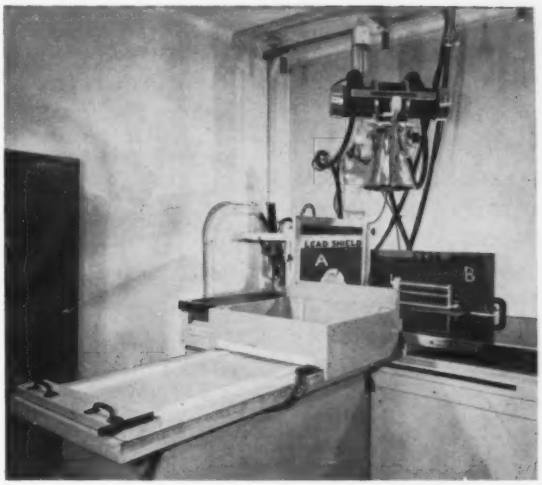

FIG. 7. Photograph showing the device applied to another table, with the lead shields in position. Shield *A* protects the physician making the injection and can be moved to any position desired. Lead cut-outs which are used on the top of the roentgen-ray table are not shown in this photograph. The wall of the hopper facing the roentgen-ray table also contains lead. The plunger for 14×17-inch cassettes is in place.

FIG. 6. Photograph showing the manner in which the apparatus is applied to a roentgen-ray table. Part *B* of the platform (see Figure 2) is inserted through the Bucky tunnel from one side of the table and is connected to Part *A* (see Figure 1) which has been brought up on the opposite side of the table.

The *stop-bars* are elevated metal rails attached by screws across the receiving end of the platform. Their purpose is to arrest the motion of the cassettes and to prevent them from sliding beyond the desired position. There are two stop-bars; one is permanently mounted and is used when 14×17-inch cassettes are employed; the other is removable, and is attached when 10×12-inch cassettes are used.

The *micro-switch* is mounted on one side of the platform, and is wired into the timing mechanism of the roentgen-ray machine. It is tripped with each back stroke of the plunger, making an automatic exposure.

Several protective *lead shields* are needed. Lead cut-outs, measuring about 2 mm. in thickness, are placed on the top of the roentgen-ray table during the procedure. A 14×17-inch or a 10×12-inch cut-out is used, depending on which size cassette is employed. An upright lead shield (Fig. 7) is attached on the far side of the roentgen-ray table to prevent scattered radiation from striking the cassettes which have already been exposed, and also to protect the technician working on this side of the table. Another upright lead shield (Fig. 7) may be placed in a suitable position across the neck or arm of the patient for the protection of the individual making the injection of contrast medium. This shield is mounted on two fairly stable legs and is adjustable in height and position. A cone is always used in order to localize the primary beam and to reduce scattered radiation.

MODIFICATION OF THE ROENTGEN EQUIPMENT

In order for this device to function satisfactorily, two slight modifications of the roentgen equipment are necessary. The first of these, the wiring of the micro-switch into the timing mechanism, has already been mentioned. (This modification is not essential if a technician is employed to activate the exposures as indicated below.) The second modification involves altering the reciprocating grid mechanism so that each exposure is made during the fast component of the movement of the grid. It should be understood that ordinarily the reciprocating grid moves rapidly in one direction, and then returns more slowly in the opposite direction. When repeated exposures are made over a period of several seconds without releasing the rotor switch (as is done in angiographic procedures), some of the exposures will occur when the grid is moving slowly. The films exposed during the slow component of the grid travel may show disturbing grid lines. This difficulty is eliminated by the insertion of an additional micro-switch into the grid mechanism in the manner indicated in the diagram (Fig. 8) and in the photograph (Fig. 9). (This switch should not be confused with the other micro-switch which is used for activating the roentgen-ray exposures). A toggle switch (also indicated in Fig. 8 and 9) is provided to bring the micro-switch in or out of the circuit, and enables the grid mechanism to be used either for conventional roentgen-ray work or for angiographic procedures.

ASSEMBLY AND OPERATION

The Bucky tray is removed from the tunnel. The platform is inserted through the tunnel, part *A* being brought up from one side, and part *B* from the other side. These two components are connected by fitted screws.

The cassettes are inspected to see that they are tightly closed and that no loose parts protrude from their surfaces. A piece of cardboard of appropriate size is tightly taped to the upper surface of each cassette, covering the entire surface. This important precaution is taken unless the upper surfaces of the cassettes are completely smooth and level. The purpose of this step is to prevent the cassettes from catching on each other and jamming the mechanism during its operation. Pieces of cardboard which come in roentgen film packages are convenient to use, are of the proper size, and are translucent to roentgen rays at the kilovoltages used.

If 14×17-inch cassettes are used, the

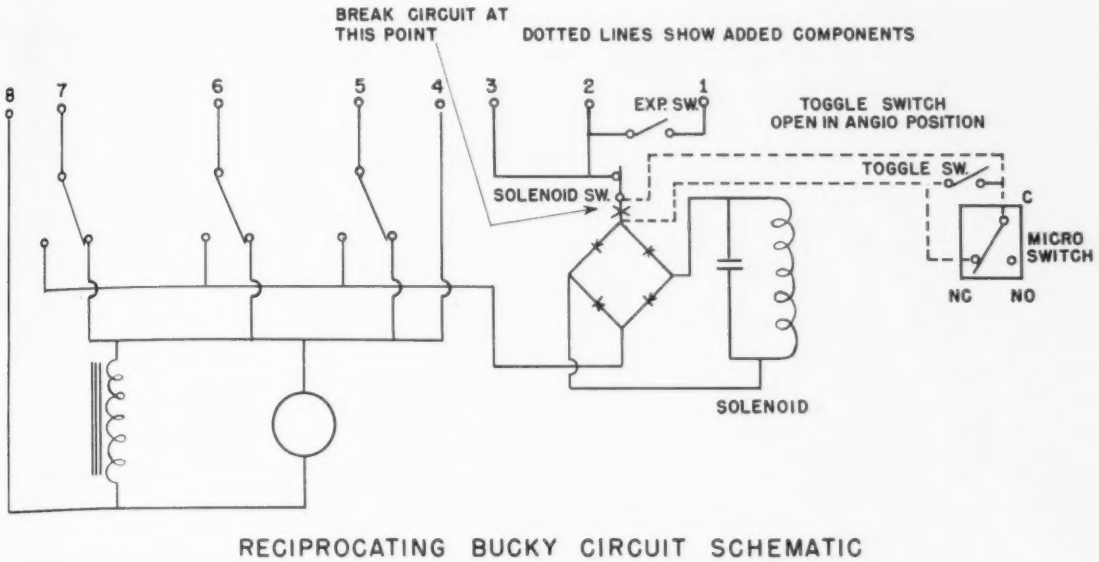

Fig. 8. Diagram of the alterations made in the reciprocating grid mechanism.

permanently mounted stop-bar is employed and the plunger designed for the 14×17-inch cassettes is placed on the platform. A pile of the desired number of cassettes is stacked in the mounted hopper. A lead cut-out with a 14×17-inch opening is placed on the roentgen-ray table. If 10×12-inch cassettes are to be used, the other stop-bar is attached in the proper position, and the plunger designed for 10×12-inch cassettes is employed. The hopper for the 10×12-inch cassettes is inserted, and is filled with the desired number of cassettes. A lead cut-out with a 10×12-inch opening is placed on top of the roentgen-ray table.

The runners of the plunger are lubricated with a little soap.

The patient is now transferred to the table. (This move can be made before the assembly of the equipment if there is any likelihood that the equipment after assembly will interfere with the moving of the patient.) The upright lead shields are placed in position. If desired, a binder for the patient's head, chest or abdomen is attached.

The bottom cassette of the pile is first pushed into position under the center of the roentgen-ray table by means of the plunger which is operated by a technician. The remaining cassettes stay in the hopper, the plunger sliding underneath the pile. A dummy or empty cassette is now

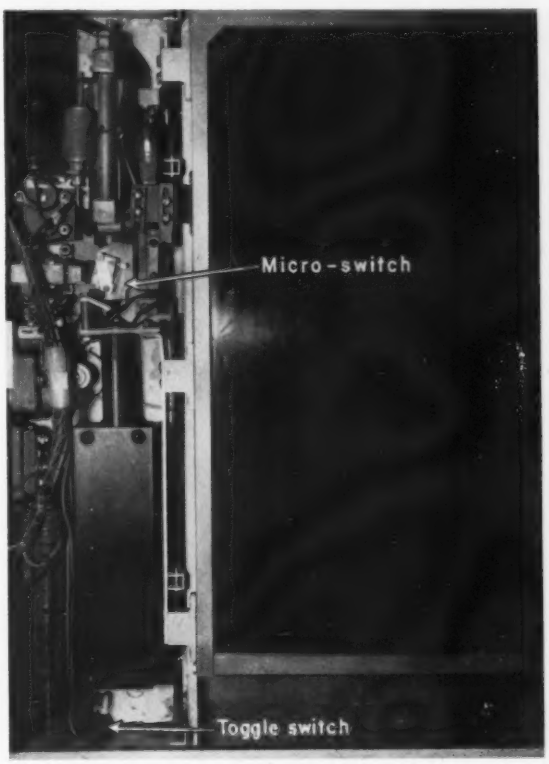

Fig. 9. Photograph showing the position of the micro-switch and toggle switch in the modification of the reciprocating grid.

placed between the cassette under the center of the roentgen-ray table and the stop-bar; there remains an open space on the platform between the dummy and the stop-bar, equivalent to the width of one cassette. The toggle switch on the reciprocating grid is set for angiographic exposures. The rotor of the roentgen-ray tube is started. The physician who is to inject the contrast medium, having previously made his preparations, now makes the injection. At the proper time he gives the signal for the first exposure. The technician operating the plunger pulls the plunger back, tripping the micro-switch and the first exposure is made. As the plunger is completely withdrawn, the entire pile of cassettes in the hopper falls down to the platform. The plunger is pushed in again, propelling the bottom cassette to the center of the roentgen-ray table. This, in turn, pushes the first cassette and the dummy along the platform until the dummy strikes the stop-bar. Sliding of cassettes beyond the desired position is thereby prevented. The dummy striking the stop-bar is immediately removed manually by a second technician. In the meantime, the plunger is again pulled back and another exposure is activated by the micro-switch. The entire operation is repeated until all of the cassettes have been pushed through the tunnel. With each stroke of the plunger, a cassette which has already been exposed

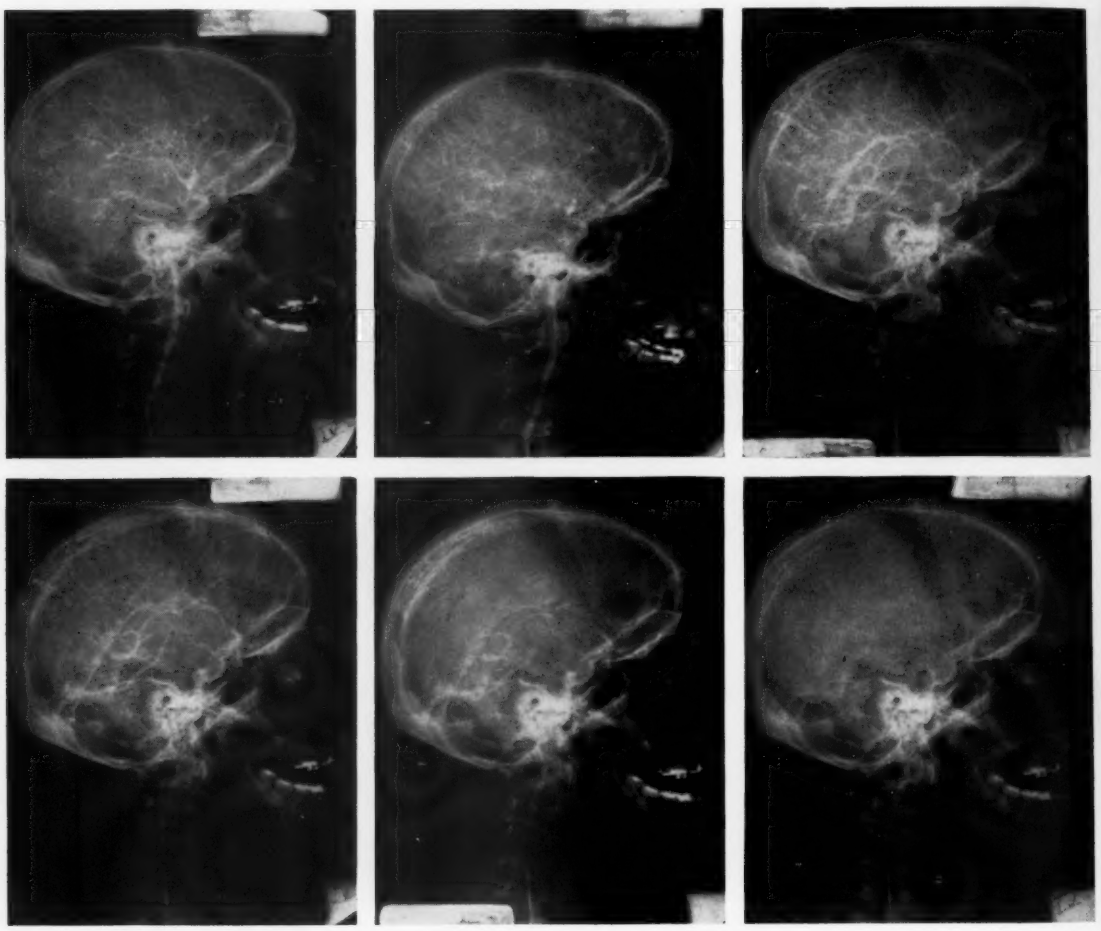

FIG. 10. Normal cerebral angiogram, lateral projection (10×12-inch films). The 6 films were exposed in six seconds. Factors: 85 kilovolts, 200 milliamperes, 36-inch target-film distance, 3/20 of a second per exposure.

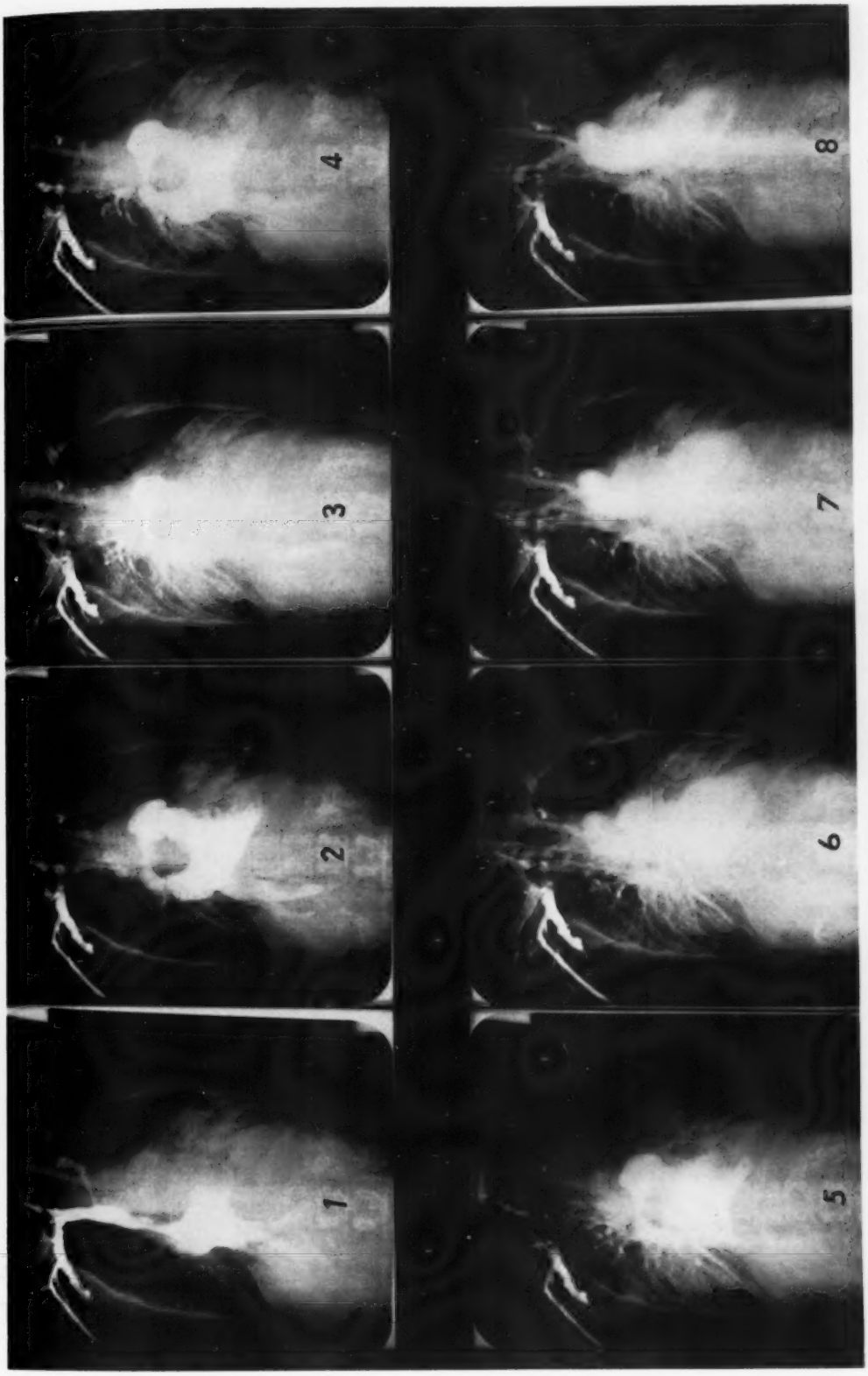

FIG. 11. Angiocardiogram (14×17-inch films) showing pericardial effusion and lack of filling of the left pulmonary vascular system. (At autopsy, metastatic lesions were found in the pericardium causing the fluid, and there was a tumor invading the left pulmonary artery with obstruction.) The 8 films were exposed in twelve seconds. Factors: 98 kilovolts, 300 milliamperes, 36-inch target-film distance, 1/30 of a second per exposure.

strikes the stop-bar, and thereby stops the cassettes behind it before being removed. The rate at which the cassettes are pushed through the tunnel is determined by the technician operating the plunger, and can be adjusted according to the needs of the particular type of examination which is being performed.

It is apparent that two technicians are needed to operate this equipment, one to operate the plunger and the other to remove the cassettes on the opposite side of the table.

Reproductions of a cerebral angiogram and of an angiocardiogram made with this device are shown in Figures 10 and 11.

If desired, an alternative method of activating the exposures may be employed utilizing a third technician instead of a micro-switch. If this method is used, the third technician activates the first exposure on signal and thereafter each time he observes the plunger being withdrawn.

DISADVANTAGES

Disadvantages in the use of this device arise from the fact that the patient must be recumbent, and the tube must be overhead. It is not possible to carry out upright angiography with this mechanism. In cerebral angiography it is necessary to shift the position of the patient's head between the anteroposterior and lateral exposures, a change which occasionally results in dislodgement of the needle from the artery. Fortunately, none of these difficulties has proved very serious as far as our own experience is concerned.

SUMMARY

An inexpensive but effective angiographic cassette-changing device is described. It is capable of handling up to twelve cassettes as rapidly as one per second; it utilizes the reciprocating grid and hence provides roentgenograms of "Bucky" quality; either 10×12 or 14×17-inch cassettes may be used; the apparatus can be adapted fairly easily to many types of existing equipment.

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Acknowledgement is made to Mr. Charles Kreutzkamp, the carpenter who built this mechanism and who supplied many of the ideas; to the members of the Medical Art Department of the University of Cincinnati College of Medicine for the preparation of the diagrams and of some of the photographs; to Miss Jane Hutzelman for the preparation of the rest to the photographs; to the Templar-Thelen X-Ray Corporation for its efforts in working out the necessary modifications in the reciprocating grid mechanism; and to Dr. Benjamin Felson, Professor of Radiology at the University of Cincinnati College of Medicine, for reviewing the manuscript.

Vol. 77, No. 2

A NEW MANUALLY-CONTROLLED PRESSURE INJECTOR FOR ANGIOCARDIOGRAPHY, RETROGRADE AORTOGRAPHY AND SELECTIVE ANGIOCARDIOGRAPHY

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THE purpose of this communication is to describe a new, manually-controlled pressure injector for use in regular angiocardiography, retrograde aortography, and selective angiocardiography performed through a cardiac catheter.

There is not now commercially manufactured in the United States a simply-operated, manually or automatically controlled angiographic injector. Models have been described, ranging from home workshop types[1] to highly complex machines working with compressed gases and capable of exerting enormous regulated pressure.[2] A model is available from Canada which works by gravity, utilizing heavy weights to press down upon the plunger of a syringe.

The equipment to be described was first conceived by the senior author in answer to a need by the surgical cardiac team of Montefiore Hospital for a pressure injector to perform retrograde aortography. Though this need appeared to be filled by the injector in early trials, its major value was found to be in a completely unsuspected field—the performance of routine venous angiocardiography.

Consistently high quality diagnostic angiocardiograms could immediately and repeatedly be produced by any physician who used the injector without previous experience. It is common knowledge among those who have attempted to introduce angiocardiography for the first time in any hospital that the major cause for failure is slow injection of contrast medium. After varying periods of experience (provided the operator's patience has not been exhausted, and the entire procedure abandoned) most physicians can be trained to inject rapidly through a 14 gauge needle and a Robb syringe the required 50 cc. of contrast medium in 2 seconds. The manual dexterity required to do this is never rapidly acquired, and initial disappointments are commonplace. Even the well-trained individual will occasionally perform a slow injection in a critical case, and a repeat examination with its attendant morbidity becomes necessary. The most important obstacle to overcome is that finally, when the individual is well-trained, he is usually (and justifiably) reluctant to continue to give injections because of the roentgenologic hazard involved.

It is for these reasons that the use of a manually-controlled pressure injector is felt to be a distinct advantage when employed for routine angiocardiography. In essence, it completely eliminates the need for experience and skill in the rapid administration of the contrast medium; the operation is reduced to manually depressing a lever. The absolute necessity for high pressure in injections for retrograde aortography and selective angiocardiography is well known and will not be discussed here.

TECHNICAL DATA

The machine (Fig. 1) consists of a base plate (1) to which are fastened a post (2) and a column (3). The post has a yoke cut into its top into which is mounted one end of the lever (4) by means of an axle passing through the yoke. The column fits through the head (5) that slides vertically

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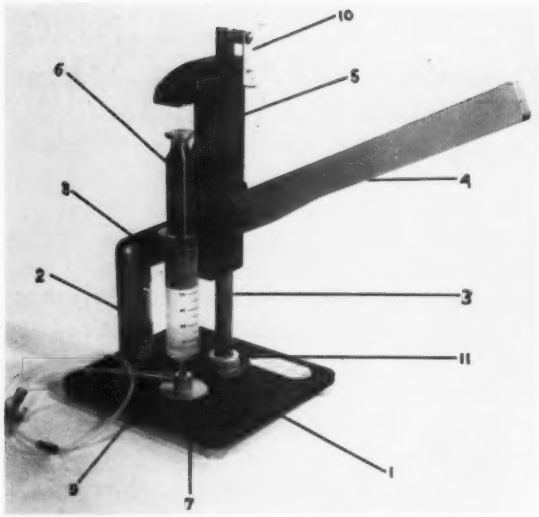

FIG. 1. Manually-controlled pressure injector, with aluminum shield removed. (1) base plate, (2) post, (3) column, (4) lever, (5) head, (6) syringe plunger, (7) pedestal, (8) syringe barrel, (9) side outlet, (10) gravity lock, (11) nylon bumper.

on it in pressing on the syringe plunger (6). In addition, there is a pedestal (7) for supporting the syringe barrel (8) under the offset section of the head. The pedestal sits in a hole in the base and has a side outlet (9) that leads to the plastic tubing and needle.

The lever, in going from the yoke to the post, slides in a closely fitted opening in the head, thus preventing the head from turning around the column, and extends to become the handle of the machine, with a mechanical advantage ratio of 4:1.

A catch (10) locks with a pin at the top of the column and holds the head and lever up automatically when the lever is elevated. It must be released to depress the lever for injection.

A fixed nylon bumper (11) fitting around the base of the column controls the final stopping position of the head so that the plunger does not touch bottom in the barrel of the syringe in use.

An aluminum shield (Fig. 2) slips over the syringe and pedestal to provide complete protection in the event of syringe breakage. The shield has an opening in its bottom edge through which connection is obtained to the side outlet of the pedestal.

Contacting parts of the machine roll on needle bearings to reduce friction. The machine attaches to any table by removable clamps (not illustrated) to permit easy portability.

DISCUSSION

The equipment described presents a number of features of clinical interest.

1. It is readily admitted that this manual injector cannot duplicate the performance of the fully automatic pressure-regulated multithousand dollar Swedish injector.[2] The latter machine provides a wide range of preselected pressures and speeds of injection. One may question, however, the value of the ability to vary pressures and speeds of injection to a fine degree. Basically, the manual injector does provide all the speeds necessary for angiographic work, a relatively slow injection for angiocardiography, and a very rapid injection for retrograde aortography and selective angiocardiography. In routine adult angiocardiography, we have standardized our procedure to the injection of 50 cc. of contrast medium in one second. To accomplish this, the lever must be deliberately, slowly depressed. Two minor advantages of this hand injector over the automatic

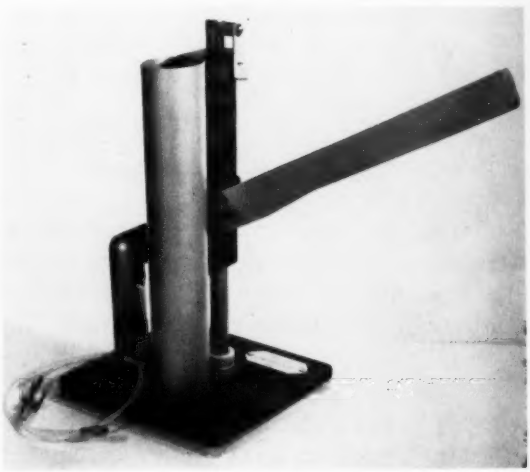

FIG. 2. Pressure injector with aluminum shield in place. Note that the syringe is now completely sealed in the event of breakage.

model are that mechanical breakdown and maintenance are no problem, and that the physician has complete control of the injection and may stop it at any time that he considers it necessary.

2. The radiation hazard of ordinary angiocardiography is greatly reduced. With the automatic pressure-controlled injector, the hazard is completely eliminated, since the machine is operated by remote control. With the manually-controlled injector, it is reduced to a negligible amount. Unlike the situation in routine angiocardiography, the operator need not remain in the room during the entire roentgen examination, attached to the syringe and the patient. Since we routinely take 30 frames with a Fairchild camera in all our cardiac studies (two frames per second), the exposure to the operator is considerable. With the manual injector, however, the operator remains in the room only during the one second required for the injection.

3. The mechanical advantage of the machine is more than the 4:1 factor resulting from the physical design. After study of the motions involved, and consideration of the comfort of the operator, it was decided to arrange the lever so that the arc travelled would begin slightly above the table top at the level of the operator's waist, and descend below the level of the table (Fig. 3). In this way, the operator not only has a convenient position for swinging the lever, but also can use his body pressure to exert further pressure at the end of the stroke. This feature is particularly desirable when high speed injections are necessary.

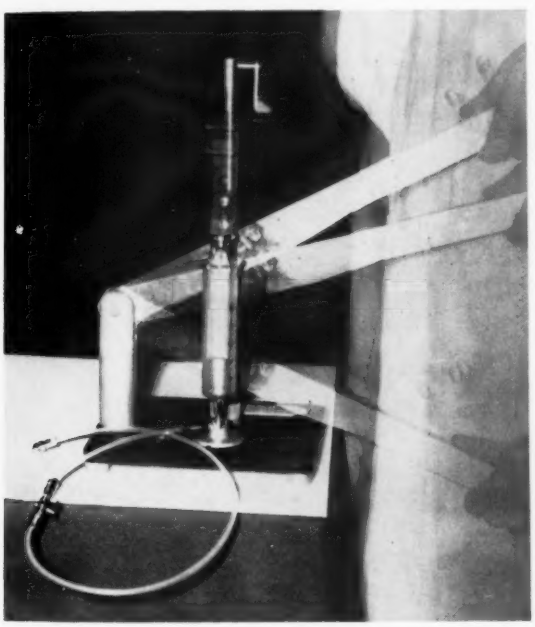

Fig. 3. The injector in action (triple exposure). The wide arc travelled by the lever extends well below the level of the table.

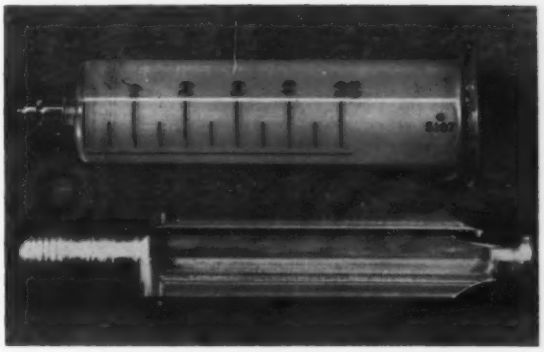

Fig. 4. Close-up of the syringe. Glass barrel with a light metallic plunger. This syringe is only needed for retrograde aortography and selective angiocardiography. An ordinary Robb syringe is completely satisfactory for regular angiocardiography.

4. The decision to use a glass syringe barrel with a metallic plunger was based upon experiment (Fig. 4). An all-glass syringe was satisfactory for routine angiocardiography, and resulted in no breakage provided that a fresh syringe was used for serial injections. When high pressures were applied (50 cc. in 0.5 sec.), it was found that the head of the plunger would shatter at the end of the thrust. The use of an all-metal syringe was considered and abandoned, since it presented the potential danger of the presence of undetected quantities of air. The optimum situation was therefore felt to be a glass barrel for visibility, with a metallic plunger to minimize breakage.

5. The aluminum shield was introduced as an additional safeguard. If a closed system is present (*e.g.*, obstruction of the lumen of the needle, or inadvertent locking

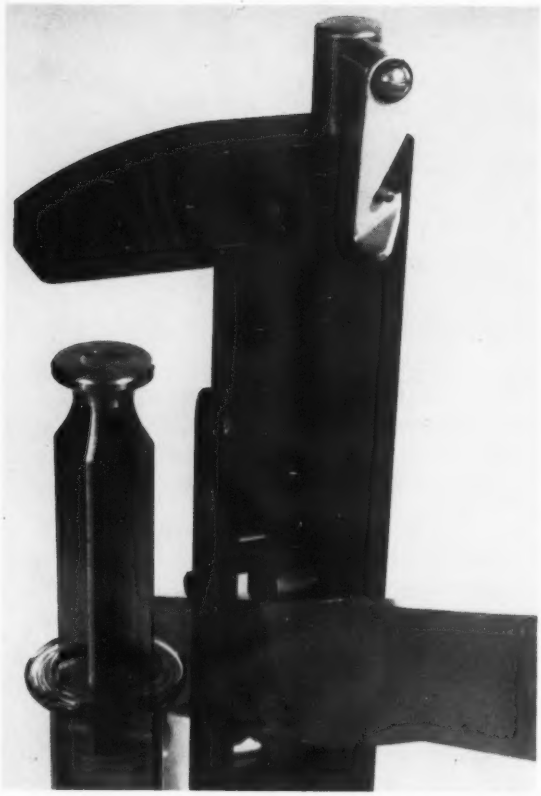

FIG. 5. Close-up of the upper end of the machine. Note the gravity lock which automatically locks when the lever is brought into the "up" position, and readily unlocks by a clockwise motion.

of the stopcock) and pressure causes the syringe to shatter, the incidence can take place without injury to either the patient or the operator. Though this had not proved necessary in our experience, it has always been reassuring to have the shield in place.

6. The gravity lock provides a convenient method for locking the lever in the "up" position (Fig. 5). When the injection is ready to be given, the lever is readily unlocked by flicking the catch over in a clockwise direction. Every time the lever is raised to the top, it is automatically locked, thereby preventing an accidental descent of the lever before the operator is ready to give the injection.

7. The nylon bumper at the bottom of the column stops pressure on the plunger well above the level of the bottom of the glass barrel. This not only prevents direct injury to the base of the glass syringe, but also prevents the introduction of any small quantity of air which might remain on top of the column of contrast medium.

SUMMARY

A new manually controlled pressure injector is introduced for angiocardiography, retrograde aortography and selective angiocardiography. Advantages of its use for routine angiocardiography are stressed. The technical features of the machine are described in detail.

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VOL. 77, NO. 2

HEMANGIOPERICYTOMA

REPORT OF A CASE

WITH SPECIAL REFERENCE TO ROENTGEN THERAPY*

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THE original description of hemangiopericytoma was published by Stout and Murray[9] in 1942. By 1954, 57 clinical cases had been reported although Stout, in a personal communication to Kay and Warthen,[7] stated that he had seen 110 examples. There has been no reference in the radiologic literature to this rare and unusual vascular tumor. A review of the available reports disclosed that only 7 patients actually had received roentgen therapy with varying dosage. The data are scant and incomplete and heretofore have not been analyzed to determine the value of irradiation.

Recently we have observed a patient with hemangiopericytoma who was subjected to roentgen therapy. The initial response was favorable. For the purpose of future reference it was thought worth while to report the evidence in detail.

PATHOLOGY

Hemangiopericytoma is a vascular tumor composed of capillaries or endothelial sprouts and of proliferating pericytes. The pericyte, originally described by the Swiss histologist Zimmermann[15] as a modified smooth muscle cell, is contractile and closely applied to the capillary wall. Its function is believed to consist in altering the size of the lumen of the capillaries. The term "pericyte" aptly describes the location of the chief constituent of the tumor. Stout postulates two prerequisites for diagnosis: (1) That the endothelia retain their normal appearance and are separated from the pericytes by a fibrous sheath and (2) that the proliferating tumor cell is the pericyte. The diagnosis is exclusively histologic. In reviewing 25 cases seven years after the original paper, Stout[12] concludes that the interim experience justifies the initial tentative classification. The histogenesis still is to be proved, but it has been possible to group similar tumors under one heading. In former years this tumor was most likely confused with the hemangioendotheliomas. These, however, are distinguished by neoplasia of the endothelia taking place within the capillary sheath. Hemangio-endotheliomas also are for the most part extremely malignant neoplasms which are blood borne and metastasize early.[11] The role of the capillaries in hemangiopericytoma is still open to debate. Begg and Garrett[4] express the opinion that the capillaries "participate in the growth only as a compensatory phenomenon." Bacon, Sherman and Campbell[3] also comment on this point.

CLINICAL FEATURES

It is not surprising that hemangiopericytoma occurs in widespread locations. Stout[12] makes the statement that the tumor may be found wherever there are capillaries. The literature records cases in which the site of origin ranges from the soft tissues of the lower extremity[5,8,9,12] to the rectum,[7] sigmoid,[2] omentum,[20] retroperitoneum,[1] extrarectal space,[3] axilla,[14] hand,[6] skin[13] and meninges.[4]

The disease may occur at any age. Stout's original paper includes 3 infants (skin lesions). Stout and Cassel's patient[10] was ninety-two years old when autopsy identified an abdominal mass present for sixty years as hemangiopericytoma of the omentum. In McCormack and Gallivan's series[8] half of the patients were in the fourth and fifth decades. They mention 8 women and

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TABLE I

SYNOPSIS OF PROVED CASES OF HEMANGIOPERICYTOMA TREATED WITH ROENTGEN THERAPY

Case No.	Author	Age, Sex	Location of Tumor	Roentgen Therapy	Results
1	Stout and Murray[9] Case No. 9	37 F	Liver metastases and rib metastases	Small doses postoperatively. No details	Relief of pain in liver area. No details regarding rib metastases
2	Sims, *et al.*[13] Case No. 2	12 M	Growth over left scapula, recurrent following electrodesiccation	Three treatments of 425 r each, 2 mm. Al filtration, 4 cm. field one month apart	Complete involution of all vascular lesions. Residual hyperpigmentation, two inches square
3	Stout[12] Case No. 19	47 F	Mass to left of uterus; residual tumor, postoperative	7,200 r (air) in twenty-four doses through multiple ports during two months	Reduced but did not completely eliminate thickening at site of the operation. No follow-up
	Stout[12] Case No. 21	11 M	Mass, left lower quadrant	a. Dec., 1935–Aug., 1936: 7,300 r (air) three fields. b. 1943: 3,000 r, anterior and posterior fields. c. 1944: 6,400 r, two anterior, posterior and lateral fields	First course: tumor shrank but did not disappear. Later courses: some relief; tumor continued to grow. Metastases to skull and femur. Death 9 years, 11 months after biopsy
5	Forrester and Houston[5]	39 M	Primary tumor, left thigh. Retroperitoneal, chest, infraclavicular metastases	"Roentgen therapy" to "mass." No details. Also had nitrogen mustard	"Little apparent effect." Died from pressure on trachea and asphyxia
6	Wise[14] Case No. 2	43 M	Large abdominal mass	No details	Five years later mass still present; was removed. Death 6 months later. General metastases
7	McCormack and Gallivan[8] Case No. 7	35 F	Retroperitoneal mass, right lower quadrant	No details	Mass receded following radiation therapy and produced no further difficulty. Death from pneumonia, 7 years later

6 men. No sex or race predilections seem to exist although most reports refer to Caucasians. The chief complaint usually is the presence of a palpable or visible mass which may be stationary or slowly growing and which may have existed for a long time before the patient sought medical advice. If it becomes symptomatic, the pain or discomfort is usually due to pressure on the adjacent tissues, especially nerves. It has no gross clinical characteristic features and the diagnosis as previously stated is made on the basis of histologic examination. McCormack and Gallivan believe that a tan color on cut section, which they found in 4 of their cases, may be a helpful means of identification. Roentgen examination gives no specific clues but is essential in detecting pulmonary or bone metastases. The presence of calcifications in the primary tumor is infrequent but is recorded by Ackerman[1] and is noted in our own case. The tumor may be self limited and pursue a benign course; it may be locally invasive or it may metastasize. Distant metastases have been described to the skin, lymph nodes, bones, lungs, liver, peritoneum and retroperitoneal space. Stout has stated that there are no criteria available that would permit one to predict whether or not the tumor is malignant. Of 110 cases, 21 per cent showed recurrence or aggressive invasion and 12.7 per cent showed metastases. It is, however, noteworthy that malignancy is displayed strikingly by tumors arising in the lower extremities and in the lower

abdomen (Stout,[12] Stout and Murray,[9] Wise,[14] McCormack and Gallivan,[8] Forrester and Houston[5]).

With the inclusion of our own experience, 10 cases of hemangiopericytoma arising in the lower extremity[5,8,9,12] are now on record. Of these, 6 have been malignant, with pulmonary metastases in 4. In 2 instances, localized recurrence was observed.

From a clinical point of view it thus appears that hemangiopericytoma is a variable entity. The prognosis depends on biologic characteristics, the location and size of the tumor, and operability. Treatment has been surgical with occasional adjuvant roentgen therapy. When local excision is successful, the prognosis is good but recurrence is still a possibility. Even in the presence of metastases there may not be any immediate danger to life and these patients may engage in productive activities for years.[2] On the other hand the disease may be rapidly fatal after metastases have appeared.

REPORT OF A CASE

History. The patient referred by Dr. Clara T. Younger was a thirty-six year old white male, a native of California and a laborer by occupation. He held jobs as a truck driver, sawman and termite exterminator. He was aware of a swelling in the right leg for the past nine years. At the onset of his condition he thought he had a "charley-horse." The leg became sore intermittently. In February, 1952 he was seen by an orthopedic surgeon (Dr. C. C. McReynolds) who noted a mass in the right leg. The maximum calf measurement on the right was $14\frac{1}{2}$ inches and on the left $12\frac{1}{2}$ inches. The minimum circumference of the right ankle was $7\frac{1}{2}$ inches and $7\frac{1}{4}$ inches on the left. The mass in the right calf measured 3 by 5 by $1\frac{1}{2}$ inches. It was fixed to the back of the tibia and not tender. Full motion of the right knee was inhibited and the right heel was stopped 3 inches from the buttock by the enlarged calf impinging upon the posterior thigh. Pulsations of the foot were normal. Roentgenograms of the leg showed a soft tissue mass but no calcifications or other distinguishing features. There was no bruit and no abnormal pulsations were palpated. No reference to skin temperature was made at that time. The lesion was diagnosed clinically as a possible fibrous (benign) mass underneath the calf group muscles and excision was advised but refused by the patient.

The patient stated that he has had "asthma" all his life. His family physician treated him for an asthmatic bronchitis in 1951, and again in December, 1953 for pneumonitis. After the acute process had subsided, he continued to complain of "pleurisy pain" in the left chest. A chest roentgenogram (Fig. 1, *A* and *B*) showed two discrete masses overlying the medial aspect of the left lower lung field. On the lateral view these masses were seen to lie anteriorly. The lesion was believed to have originated in the anterior mediastinum and was considered to be a dermoid or teratoma or a tumor of pleural or pericardial origin, although the possibility of metastatic malignancy was suggested.

The chest findings presented the main diagnostic and therapeutic problem. The fact that the patient also had an enlargement of the right calf was discounted at first since it had been present for many years and was thought to be benign. However, inasmuch as metastatic malignancy could not be excluded, it was decided to perform a biopsy on the potential primary lesion. The diagnosis of hemangiopericytoma of the leg was made; yet there still existed some doubt as to whether the mass in the chest was metastatic or not. As an apparent localized lesion, it probably was operable and exploration would be of material assistance in planning therapy for the leg. It was reasoned that if the chest lesion was an independent entity and resectable, then the tumor of the calf could be attacked vigorously. On the other hand, definite proof of metastases would militate against a radical procedure such as amputation.

Physical Examination. On admission to the St. Francis Hospital on March 23, 1954, the patient was in no apparent distress. His family history was noncontributory. His past history was outlined above. His smoking habits consisted on one pack of cigarettes a day. Examination showed a few small lymph nodes in both axillae and inguinal areas. The chest was clear to auscultation and percussion. No palpable abdominal masses were noted. The circumference of the right calf was $16\frac{3}{4}$ inches and of the left calf $12\frac{3}{4}$ inches.

Laboratory Findings. Urinalysis was negative.

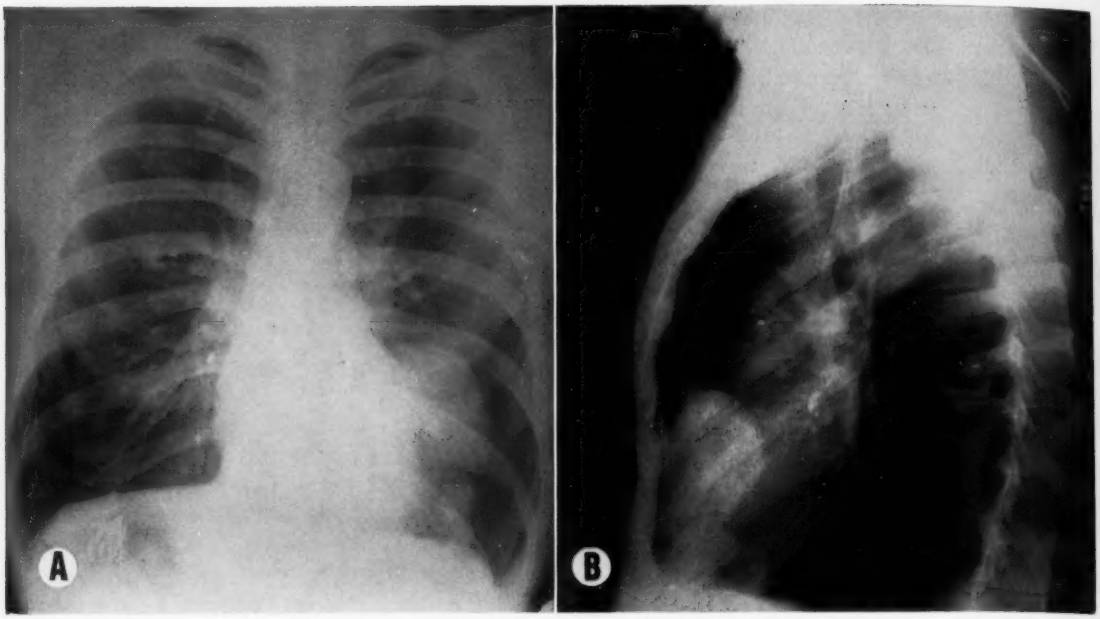

FIG. 1. March 12, 1954. (*A*) Posteroanterior roentgenogram of the chest showing a mass in the left lower lung field. (*B*) Left lateral view discloses that the mass consists of two discrete densities in the anterior chest.

The blood count showed the following: Hemoglobin 14.2 gm. per 100 cc., erythrocytes 4.7 million, leukocytes 13,400, neutrophils 84 per cent (segmented cells 80 per cent, band cells 4 per cent), eosinophils 2 per cent, lymphocytes 12 per cent and monocytes 2 per cent. The red blood cells and platelets appeared normal. The Kline test was negative.

FIG. 2. March 23, 1954. Soft tissue mass in the calf muscle of the left leg.

Roentgen Findings. Chest roentgenograms of March 12, 1954 (Fig. 1, *A* and *B*) showed two well defined homogeneous masses in the left lower lung field anteriorly. The larger one measured approximately 6 by 7 cm. at its greatest dimension and the smaller one, distal to it, 3 by 5 cm. No other lesions were identified with certainty at that time.

Films of the right leg (Fig. 2) dated March 23, 1954 revealed a homogeneous soft tissue mass involving the entire calf. It measured approximately 10 by 20 cm. on the lateral view. Heavy penetration films (Fig. 3, *A* and *B*) showed increased medullary trabeculation with slight central rarefaction in the upper shaft of the tibia extending over an area 3 by 5 cm. in diameter. The posterior cortex of the upper tibia was somewhat striated in appearance but there was no evidence of invasion or breakthrough. However, the tumor had produced a definite pressure defect on the posterior middle tibia and medial cortex of the fibula as evidenced by some localized thinning. There were also a few specks of calcium deposits on the medial side of the tibia where the cortex formed a small rounded prominence. The bone changes were believed to be secondary in nature.

Survey roentgenograms of the skull and

Fig. 3. March 23, 1954. Overpenetrated films of the left leg. (*A*) Increased medullary trabeculation in upper shaft of tibia with slight central rarefaction. Pressure defect, medial cortex of fibula. (*B*) Lateral view. Pressure defect on posterior tibia.

pelvis failed to show any evidence of metastases.

Operative Findings. Biopsy of the right leg (March 24, 1954) showed the following: There was a large tumor mass underneath the calf muscles. A portion of the tumor exposed was just medial to the tibia and was firmly attached to it. It was glistening and white in color and appeared to be fairly well encapsulated.

Left thoracotomy by Dr. William Henry on April 3, 1954 revealed two rounded tumor masses composed of pale gray, slightly hemorrhagic tissue in the most dependent portion of the lingular segment of the left upper lobe. They were adjacent to each other. The larger tumor was adherent to the pericardium and to the left phrenic nerve. Scattered throughout the left lung were numerous small nodules, at least seven of which could be palpated. The lesions in the lingular segment of the left upper lobe were dissected away from the pericardium with slight difficulty. The dependent portion of the lingula containing the tumor was then excised. A 1 cm. lesion of the left lower lobe was also removed by the wedge technique.

Pathologic Report (Dr. D. J. Rehbock). The specimen removed from the leg on March 24, 1954 was a narrow, wedge shaped biopsy 2 cm. in length. It was composed of tumor covered over one surface by a thick fibrous capsule.

The specimen from the lung removed on April 3, 1954 consisted of two separate masses, one weighing 28 gm., the other 156 gm. Each consisted of tumor with a small amount of attached lung tissue partly covered by pleura. Each tumor had an external surface which was lobulated and covered by a thin fibrous capsule through which small tumor excrescences protruded. The cut surface was a mottled pale gray, yellow and red with friable, cellular tumor tissue interspersed with numerous large, irregular

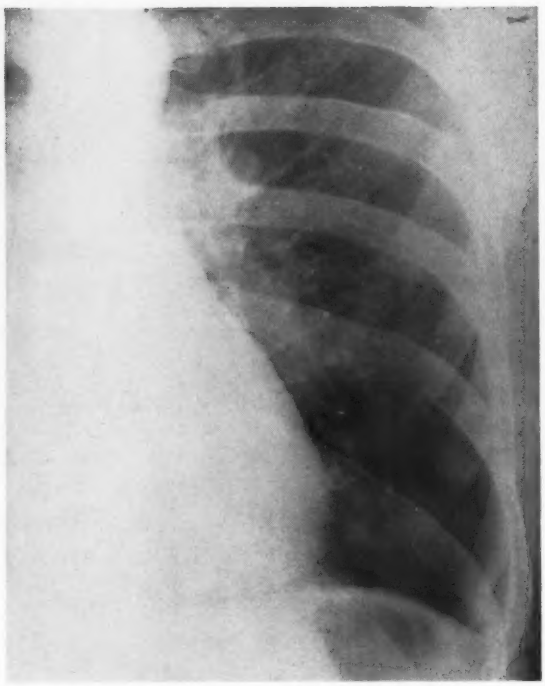

FIG. 4. June 16, 1954. Nodular metastases shown in the left lower lung field. Note distinct nodule in left hilum. Metastases were present in both lungs.

vascular spaces filled with fluid blood. The larger spaces measured as much as 1.5 cm. in diameter.

Microscopic examination showed the same histologic picture in the leg and pulmonary tumors (Fig. 6). The tumor cells were of medium size with rounded or irregular vesicular nuclei which tended to be centrally placed and were surrounded by a moderate amount of pale, vacuolated cytoplasm. Nucleoli were small and inconspicuous and mitotic figures were not seen. The cells were compactly arranged with no visible stroma except for numerous capillaries and larger vascular structures. Tumor cells were closely applied to the external surface of the capillary endothelium.

Postoperative Course and Roentgen Therapy. The patient made an uneventful recovery from the surgical procedures. Deep roentgen therapy to the right leg was started immediately (April 8, 1954) and was continued on a five day per week schedule until April 30, 1954. The leg was crossfired alternately through one medial and one lateral port, 20 by 20 cm. in size, at the daily rate of 300 r (air). Each field received 2,400 r (air). This resulted in a tumor dose of 4,440 r at 6 cm. The physical factors were as follows: 250 kv., 15 ma., 0.5 mm. Cu and 1 mm. Al filtration, target skin distance 50 cm. and half-value layer 1.5 mm. Cu.

There was a slight erythema at the completion of therapy. By May 10, a moist radiation reaction was fully developed and was completely healed by June 16. There was loss of hair and some pretibial edema with slight pitting of the skin. The circumference of the leg still measured 16½ inches but the calf was much softer and the patient positively stated that his leg no longer felt "stony hard" and that the sensation of tension, present since the onset of his condition, for the first time had disappeared. By July 27, a definite increase in skin temperature was noted. The leg had not become smaller in size (16½ inches) but measured about one inch less upon arising. Also the pretibial and ankle edema had remained unchanged.

Pulmonary metastases were obvious on a chest roentgenogram taken on June 16, 1954 (Fig. 4). It showed multiple nodular lesions in both lungs, especially in the lower lobes, measuring from 5 to 12 mm. in diameter. The left hilum was distinctly enlarged by metastatic invasion. Subjective complaints consisted chiefly of pain in the left chest, malaise and some weakness. It was then decided to selectively irradiate the left lung and to leave the lesions on the right side as a control in order to determine the degree of radiosensitivity of the tumor. Accordingly, the left lower lung was treated through one anterior and one posterior 15 by 15 cm. field with the dome of the diaphragm, as determined fluoroscopically, as the lower border. Physical factors were the same as above. Each day 300 r (air) were administered for a total dose of 2,100 r (air) to each field. The calculated tumor dose was 2,163 r at 10 cm. The nipple was protected by lead. A moderate skin reaction ensued. The patient was ambulatory throughout the course and was able to work part time. By August 26 (Fig. 5), the nodules in the left lung had disappeared while there was no discernible change on the right side. Films at monthly intervals essentially presented the same findings. Four months after completion of therapy no metastases could be detected in the left lung. The nodular lesions in the right lung had not grown any further but there was a slight bulge in the right upper mediastinum, suggesting lymph node involvement. Roentgenograms of the right leg were comparable with the preopera-

tive and pre-irradiation films. The skin temperature was returning to normal; only the edema persisted. On November 2, the right leg measured 16 inches in circumference; the measurements of the right ankle were 10 inches and of the left ankle 8 inches. At that time the right lung had not been treated. The patient was still working as a termite exterminator.

Soon afterwards his condition began to deteriorate rapidly. A hard mass appeared in the right parietal area of the skull. A chest roentgenogram showed overwhelming metastases throughout the right lung and left upper lobe. Fewer lesions were seen in the irradiated left lower lobe. The patient became increasingly anemic and dyspneic and died on February 3, 1955.

Autopsy Findings (Dr. D. J. Rehbock). The autopsy report is condensed and limited to pertinent findings.

External examination showed irregular pigmentation over the calf of the right leg which was slightly larger than the left. There was some brown pigmentation over the lower portion of the left thorax.

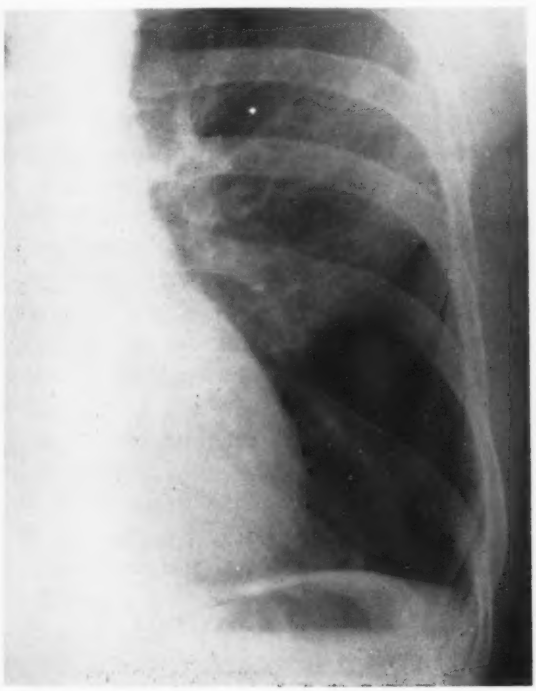

Fig. 5. August 26, 1954. Metastases in left lower lung field have disappeared following roentgen therapy. The nodule in the left hilum is no longer visible.

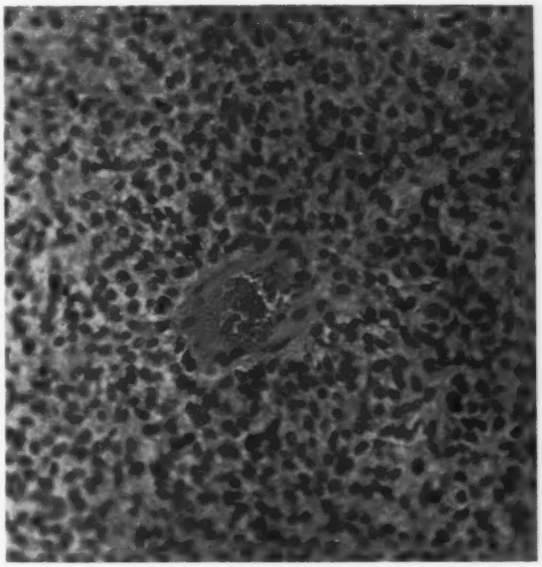

Fig. 6. Photomicrograph of tumor metastasis in lung. Tumor cells are closely applied to the external surface of the vessel wall (×360).

On section the peritoneal cavity was free of fluid. The liver extended well below the level of the umbilicus. The right pleural cavity contained approximately 300 cc. of thin, blood tinged fluid. The pleural surfaces were studded with large tumor masses measuring up to 3 cm. in diameter, lobulated masses projecting from the parietal pleura as well as from the visceral pleura. In some areas there were adhesions which bound lung and tumor to the chest wall. No free fluid was present in the left pleural cavity but there were fibrous adhesions between the entire left lung and the chest wall.

The weight of the lungs together was 3,850 gm. The right lung showed many large tumor masses projecting from the pleural surface and involving the lung parenchyma with distortion of the lobes and fusion of the interlobar spaces. The tumor over the pleura of the lower lobe measured up to as much as 3 cm. in thickness. It was white, opaque, cellular and friable. The left lung showed irregular pleural invasion and scattered tumor nodules within the parenchyma. The tumor was much less extensive than was seen on the right but on the cut surface it had the same white, cellular appearance. There were massive bronchial lymph node metastases and there was involvement of the inner surface of the sternum with no evidence of invasion of the bone.

The liver weighed 1,520 gm. In the left lobe

there were several scattered, small nodules, the largest 0.8 cm. in diameter. None were seen in the right lobe.

The retroperitoneal lymph nodes were small, the largest one 1.5 cm. in length but they were firm and showed tumor growth in the lower abdominal portion adjacent to the aorta.

On removal of the calvarium there was found a large area, 10 cm. in diameter, in which there was bony destruction involving the right parietal and occipital bones. The underlying dura was adherent to this area and projections of tumor extended from the dura into the bone as well as into the subdural space. The tumor had a nodular surface and it was elevated as much as 1 cm. in some places. The inner and middle tables were affected with no change in the outer table except in the midportion where there was a mass, 2 cm. in diameter, which projected outward beneath the scalp. Over the left parietal lobe there was one small area, 1 cm. in diameter, where the tumor had spread to the dura and the inner table of the skull.

Section of the calf of the right leg revealed a well circumscribed tumor mass 10 cm. in diameter, which involved the deep musculature but which appeared to lie between fascial planes without any irregular invasion of surrounding muscle. The cut surface showed moderately firm, friable areas which were pale brown and semi-translucent, other areas being cystic and occupied by blood clots. There was a moderate amount of fibrosis around and extending through the tumor in irregular pale gray fibrous strands.

Microscopic Findings. A section taken from the periphery of the right lung showed a highly cellular tumor, the cells being characterized by small, round or slightly ovoid nuclei surrounded by a relatively large amount of clear cytoplasm. These cells had no architectural pattern and they were supported entirely by a delicate vascular stroma with numerous small capillary vessels. The capillary vessels were lined by endothelium and were immediately surrounded by tumor cells. The pleural surface was covered by a thick layer of organizing fibrinous exudate. A section taken from the periphery of the left lung showed tumor of the same type as described above, the cellular structure being identical. The tumor involvement was less extensive.

A section of liver showed one metastatic tumor nodule of the same type as described above.

A section of the involved dura showed highly cellular tumor of the same type as described above projecting from both surfaces. Tumor was present within vascular channels within the fibrous membrane.

Five different sections taken from the right leg showed most of the tumor to be necrotic and represented only by granular acidophilic material containing no cellular structures except for a few small blood vessels. In some areas there was visible tumor and it had the same cellular structure as that described above. In these areas there were foci of hemorrhage and there were numerous large channels filled with blood, the channels being lined by neoplastic cells. Hemosiderin pigmentation was present within the degenerated areas in some places.

Final Pathologic Diagnosis. 1. Malignant hemangiopericytoma of the right leg with extensive metastases to lungs, pleura, intrathoracic and abdominal lymph nodes, dura mater, bony calvarium and with a few small metastases to the left lobe of the liver. 2. Hydrothorax, right. 3. Emaciation.

DISCUSSION

The pattern of evolution of metastasizing hemangiopericytoma was characteristic in the above case. There were several features identical with those exhibited in 4 patients described in the literature[5,8,9] in whom the primary tumor originated in the leg. All were in the younger age group (twenty-seven to forty). The tumor was known to have existed for several years. All developed pulmonary metastases and 3 out of 4 died from the disease.

An analysis of Table 1 reveals that there are seven verified cases of hemangiopericytoma on record that have received roentgen therapy. The physical factors, dosage and number and size of ports are either incomplete or missing altogether. High voltage radiation probably was employed in all patients except one (Case 2) who had superficial therapy. Cases 3 and 4 appear to have had an adequate dose administered although the time interval in

the first course in Case 4 seems to be unduly long (December, 1935–August, 1936). The results were as follows: Complete involution of all vascular lesions was achieved in a recurrent skin tumor (Case 2) with three treatments of 425 r, one month apart. This was a remarkably good result with a relatively small dose. In Case 7 a retroperitoneal mass receded after roentgen therapy and the patient lived for seven years without relapse, death being due to other causes. In 2 instances the tumor became smaller but one patient (Case 4) who lived for about ten years afterwards, eventually succumbed to the disease and there was no follow-up in the other one (Case 3). Case 6 presumably was helped by irradiation because he lived for five years after exploration and roentgen therapy. Palliation was mentioned twice (Case 1 and 4). It was stated only once (Case 5) that "radiation had no apparent effect." Unfortunately no details were given.

Thus in 6 out of 7 cases, roentgen therapy exerted a beneficial influence ranging from palliation to disappearance of the lesion. Our own findings demonstrated a softening of the primary tumor with relief of tension and discomfort. The most dramatic proof of radiosensitivity, however, was the disappearance of the pulmonary metastases that were treated with roentgen therapy, whereas the untreated lesions were not affected. Furthermore, microscopic examination of the autopsy specimen of the primary lesion in the leg showed a marked necrosis with only a few viable tumor cells remaining. There was also much less involvement of the irradiated left lung as compared to the chaotic tumor growth in the right chest.

It was of interest to note that roentgen therapy produced only a softening of the primary tumor but no significant decrease in the size of the leg. The skin temperature remained elevated, indicating persistent augmented vascularity. Evidently, as late as three months after treatment, there was still an abnormal increase in blood supply. This may have been due to reactive hyperemia following deep roentgen therapy. However, the question also arose whether the capillary proliferation responsible for the higher temperature was merely a concomitant feature of the tumor as suggested by Begg and Garrett[4] rather than the neoplastic process itself.

No dogmatic generalization can be made regarding the effects of irradiation on hemangiopericytoma on the basis of a single observation; however, the results of the planned course of therapy in our patient corroborate the experience of others in that at least palliation can be obtained. It is possible that the tumor is quite radiosensitive requiring smaller doses than those administered. In the majority of cases treated, the lesion proved to be responsive. A study of the preceding case histories leaves the impression that roentgen therapy might have been of value at times.

There has been an increasing number of new cases since 1949 as compared to the isolated reports between 1942 and 1949. Even though the tumor is admittedly rare, it is to be expected that hemangiopericytoma will be recognized in the future more often. With regard to management, the following suggestions are made: The primary treatment of hemangiopericytoma is surgical removal. Postoperative roentgen therapy is indicated when excision is impossible or incomplete or when there is a local recurrence. A single metastasis (*e.g.*, in the lung) can either be treated by resection or irradiation. In the presence of multiple metastases palliative irradiation should be administered. There is no reference to any experience with preoperative roentgen therapy. It might be useful in the highly vascular lesions, especially when located in the pelvis or retroperitoneal space.

SUMMARY AND CONCLUSION

1. A case of primary hemangiopericytoma arising in the soft tissues of the leg with generalized metastases is reported.

The pathologic and clinical features of the disease entity are discussed. Autopsy findings are included.

2. Objective response of the tumor to roentgen therapy is demonstrated. This supports the experience in 6 out of 7 patients previously described in the literature.

3. It is suggested that irradiation is a valuable adjunct to surgery in the treatment of this rare tumor.

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EDITORIAL

RADIATION PROTECTION IN ANGIOGRAPHY

THE "maximum permissible dose" of ionizing radiation is continually being revised downward.[9] This trend will doubtless continue. Recent scientific and political activities directed at assessing the hazards of radiation for the world population, particularly future generations, have brought the diagnostic medical applications of roentgen rays under the closest scrutiny.[1,2]

The utilization of angiographic procedures—angiocardiography, aortography, cerebral angiography, and peripheral arteriography—is rapidly expanding. In this field, and most prominently in cardiac studies, exploitation of the possibilities of ever increasing numbers of roentgen film exposures in rapid sequence has kept pace with technical developments aimed at reducing radiation dose. Thus, relatively large local doses are frequently administered and an arbitrary "maximum permissible" exposure to the patient* may be approached or exceeded in a single rapid sequence angiographic study. In such procedures the very real hazard of overexposure of the medical personnel involved must always be considered and controlled.[5,6,7]

The clinical situation usually fraught with the greatest radiation hazard is the complicated cardiac diagnostic problem especially when evaluation of possible congenital defects is required. The patient, commonly an infant or child, may go through a succession of examinations including fluoroscopy and chest roentgenography, the number of which is often directly proportional to the remoteness of the family from the "larger center" where the definitive study and decision as to treatment are ultimately effected. At each stage in this diagnostic work-up the patient's physician and the radiologic consultant have the joint responsibility of considering whether more elaborate studies in their particular locality or institution are justified. It must be emphasized that, for the patient, the other risks encountered in heart catheterization or angiography overshadow the radiation hazard. However, such a complete study may result in a total skin dose of well over 100 r; often such a combined study is accomplished in a single day. (First degree erythema may result from approximately 150 r diagnostic quality roentgen rays delivered in one day; if such a dose is given no additional roentgen ray exposure should be permitted for fourteen days.)[7]

Fluoroscopy during heart catheterization retains greater radiation danger than the usual angiographic procedures. The best modern fluoroscopes deliver 6 to 8 r per minute (3 ma.—85 kv.) at the table top but, unfortunately, many machines still in use produce very much larger skin dose rates. The catheterization procedure may involve ten to twenty minutes of fluoroscopic time. As in any radiologic technique, the field size should be kept at a minimum with the beam adequately coned (direct radiation need reach the gonads in relatively few angiographic procedures). Filtration of at least 2 mm. Al added is essential, and the maximum practical target to skin distance should be used. The total fluoroscopic time permitted must be regulated by the known r output of the apparatus used.

Practical fluoroscopic image intensifiers are now available which make possible reduction of the fluoroscopic dose by one fifth to one tenth with improved visualization.

* A number of authorities have used approximately 25 r for this dose.[3,4]

Substitution of image intensifier fluoroscopy for conventional screening can now be strongly recommended for all cardiac fluoroscopy in infants and children and for all heart catheterization.[8]

For angiography direct roentgenograms exposed in rapid sequence remain most generally applicable at the present writing. Photofluorography with the best available mirror optics requires 5 times the dose needed for an equivalent direct roentgenogram. It has been well emphasized that image quality rather than the number of films exposed per unit of time is a prime consideration. However, the image intensifiers permit cineangiography at high speeds (in excess of 60 frames per second) with doses from 1 to 5 per cent of those previously required for direct motion picture photography of the fluorescent screen.[8] This renders practical motion picture angiocardiography of infants, with good projected image quality, at dosage levels less than required for conventional fluoroscopy. The advantages of motion study may well outweigh the disadvantage of poorer single frame image quality in comparison to direct roentgenography. The image intensifiers should find a place in selective angiocardiography in adults in addition to their proved and obvious utility in pediatric cardiology.

The responsibility of the radiologist lies in knowing the magnitude of the radiation hazard and in minimizing it for the patient and for the medical personnel exposed, including radiologists, surgeons and anesthetists. This requires monitoring not only of the apparatus used but also of actual procedures. The safest available roentgenologic technique which can be expected to give the needed information should be employed.

By institution of well known measures angiographic examinations of any kind can be made safe for medical personnel. For the patient the radiation exposure, when kept to a practical minimum, is a negligible price to pay for the possible benefits from an important diagnostic study judiciously undertaken and competently performed.

Herbert M. Stauffer, M.D.

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SOCIETY PROCEEDINGS AND NEWS ITEMS*

LOS ANGELES RADIOLOGICAL SOCIETY TO SPONSOR NINTH ANNUAL MID-WINTER RADIOLOGICAL CONFERENCE

The Ninth Annual Mid-Winter Radiological Conference, sponsored by the Los Angeles Radiological Society, will be held at the Biltmore Hotel, Los Angeles, California, Saturday and Sunday, February 23 and 24, 1957.

An outstanding program of pertinent interest has been arranged, and the guest speakers will be Dr. John Caffey, New York; Doctor Johan Frimann-Dahl of Ulleval Hospital, Oslo, Norway; Doctor Merrill C. Sosman, Boston; and Professor Brian W. Windeyer, University of London and Middlesex Hospital, London, England.

The conference fee of $20.00 includes two luncheon meetings featuring questions and answers. A banquet ($7.50 per plate), preceded by cocktails will be held Saturday evening. Reservations may be made through Dr. Louis J. Bonann, 1245 Glendon Avenue, Los Angeles 24, California.

Courtesy cards will be available to residents in radiology and radiologists in the Armed Forces by advance registration, with reduced tariff for the luncheons and banquet. Hotel reservations should be made promptly through the Convention Manager, Biltmore Hotel, Los Angeles, California.

EASTERN CONFERENCE OF RADIOLOGISTS TO BE HELD IN NEW YORK CITY

The Eastern Conference of Radiologists is to be held at the Waldorf-Astoria Hotel, New York City, March 7, 8 and 9, 1957. Invitation is extended to all radiologists and their families to attend the Conference. A theater party has been arranged for Thursday evening, March 7, following the traditional cocktail party. The price of tickets on the mezzanine is $5.75, while orchestra seats are $7.50 and $6.25. The Conference registration fee for physicians and visitors will be $25.00. The fee for wives and guests will be $15.00. Checks covering registration fees and theater tickets should be mailed to Dr. Harold G. Jacobson, Treasurer, Department of Radiology, Montefiore Hospital, 210th Street and Bainbridge Avenue, New York, New York.

DR. JOHN CAFFEY DELIVERS MACKENZIE DAVIDSON MEMORIAL LECTURE

Dr. John Caffey, Columbia-Presbyterian Medical Center, New York City, delivered the Mackenzie Davidson Memorial Lecture and received the medal and award of the British Institute of Radiology at its annual meeting in London, England, December 6 and 7.

THE AMERICAN BOARD OF RADIOLOGY

Candidates who started their residency in 1956 will be expected to devote three years to formal residency training in an approved department of Radiology, *plus* one year of practice or additional training in order to be eligible to appear for examination by the American Board of Radiology.

Those individuals who may be called to military duty during their residency and have been assigned to a department of Radiology for one year or more, may substitute this time for the required one year of practice or additional training.

All candidates for examination after 1956 must have received *three* years' formal, approved residency training regardless of military duties.

DR. B. R. KIRKLIN, *Secretary-Treasurer*
Rochester Minnesota

* The list of Meetings of the Radiological Societies appears in the January, 1957, issue of the JOURNAL, and will be published in the March number.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Spléno-Portographie. Etude Radiologique et Clinique de la Circulation Portale Normale et Pathologique. Exploration des Organes Sus-Mésocoliques. Par Lucien Leger. Paper. Price, 1.600 fr. Pp. 166, with 82 illustrations. Masson et Cie, Éditeurs, 120 boulevard Saint-Germain, Paris 6e, France, 1955.

Contrast visualization of the spleen, splenoportal venous system and liver by intrasplenic injection of opaque material constitutes a recent development. This book presents a series of surprising and diverse roentgenologic documents. It is intended to show the conclusions which may be drawn from a morphologic study of the splenoportal system, including the spleen, the pancreas and the liver. The illustrations are exceptionally good. A final chapter of the book deals with insufficiencies and limitations, as well as with hopes for the future of splenoportography. As a reasonable precaution, the patient should be under observation in the hospital for twenty-four hours. Repetition of the maneuver after an interval of a few days or weeks causes no inconvenience. The author reports a case where the test was made four times without harm, and he cites a case of hepatic cirrhosis where the test was done eight times in fourteen months without any accident or untoward symptoms.

James T. Case, M.D.

Clinical Pathology; Application and Interpretation. By Benjamin B. Wells, M.D., Ph.D., Director of Clinical Investigation, The Lynn Clinic, Detroit. Former Professor of Medicine and Chairman of the Department of Medicine, Creighton University School of Medicine, Omaha. Second edition. Cloth. Price, $8.50. Pp. 488, with 25 illustrations. W. B. Saunders Company, 218 West Washington Square, Philadelphia 5, Pennsylvania, 1956.

The selection and interpretation of laboratory tests as aids in clinical diagnosis are matters which have rapidly increased in complexity. For the practicing physician, faced with a bewildering array of possible examinations, this book offers a synopsis of generally available laboratory procedures and gives a brief summary of their merits and limitations. It is not a textbook of clinical pathology nor does it claim to be one. The new edition has added brief discussions of the *C*-reactive protein test, the transaminase test, and a chapter on laboratory procedures. The text has been rearranged and the format considerably improved.

In the reviewer's opinion this book could benefit greatly from the addition of a bibliography. Its coverage of subjects is of necessity somewhat superficial, but the more inquisitive reader finds almost no references to aid him in a further pursuit of a given topic. It fulfills its announced intent of providing a survey of the expanding field of clinical pathology, but it could be infinitely more valuable if future editions were supplemented by many more pertinent, up-to-date references. The subjects of uterine exfoliative cytology, paper electrophoresis and chromatography might also merit some mention. However, the author's wide experience and comprehension of the uses and pitfalls of laboratory examinations make this a useful reference book for the busy physician.

Edwin M. Knights, Jr., M.D.

Atlas of Exfoliative Cytology. Supplement 1. By George N. Papanicolaou, M.D., Ph.D., Clinical Professor of Anatomy Emeritus, Cornell University Medical College. Looseleaf. Price, $4.00. Sixteen plates. Published for The Commonwealth Fund by the Harvard University Press, Cambridge, Massachusetts, 1956.

As stated in the preface to the atlas, published in 1954, the loose-leaf style was adopted in the hope of keeping abreast of new advances through periodic addition of illustrative and descriptive material. This, the first supplement, follows the format and style of the atlas. It includes 16 new full-page plates in color on the following topics: female genital system, respiratory system, digestive system, exudates, breast, miscellaneous, and mitosis. A subject index to the supplement is also provided. In 14

of the 16 plates comparative illustrations are given of histopathologic and cytologic appearances, "thus bringing out clearly not only similarities but also dissimilarities, which are often marked." This is the principal feature of the supplement and represents its main improvement over the original material. The illustrations are of surpassing quality and are accompanied by brief descriptions and discussions of each figure.

With time and the experience of many workers, we may expect to have presented for us additional supplements in which the definition of cellular morphology is constantly rendered sharper.

S. E. Gould, M.D.

Ankylosing Spondylitis. By J. Forestier, M.D., F.A.C.R. (Hon), Aix-les-Bains; F. Jacqueline, M.D., Aix-les-Bains; and J. Rotes-Querol, M.D., Barcelona. Translated by A. U. Desjardins, M.S., M.D., F.A.C.P., F.A.C.R. Cloth. Price, $10.75. Pp. 374, with 145 illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1956.

The authors used 40 cases with an evolution of twenty years, as a group, to compile the incidence of the disease, the symptoms, roentgen findings, clinical forms, and pathology, and to evaluate their success in the treatment of the disease. Their personal observations and their conclusions are compared impartially with those of other authors.

The volume is easy to read and to handle. The illustrations are above average. A very comprehensive bibliography, covering both historical and clinical phases of the subjects discussed, is included. This book will be valuable to those interested in this phase of rheumatology, including its roentgenologic characteristics.

Frank P. Walsh, M.D.

Roentgen Interpretation of Fractures and Dislocations. By Joseph Levitin, M.D., Chief, Department of Radiology, Mount Zion Hospital, San Francisco, California; and Ben Colloff, M.D., Associate Chief, Department of Orthopedic Surgery, Mount Zion Hospital, San Francisco, California. Cloth. Price, $7.75. Pp. 284, with 258 illustrations. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1956.

The authors, a roentgenologist and an orthopedic surgeon, have successfully collaborated in presenting in straightforward, easily understandable prose a basic concept of what constitutes a generally satisfactory reduction. Two hundred and fifty-eight diagrams well illustrate the mechanism of fracture with associated muscle pull. The chapters on the pelvis, thorax, and spine are, however, rather sketchily outlined.

The authors properly stress the vast difference between a perfect anatomic alignment and a good functional result. These are not necessarily synonymous. The functional restoration must be constantly emphasized.

Roentgenologists are commonly required to determine the onset of healing. The authors evaluate the factors which delay healing: distraction due to skeletal traction, use of hanging casts, inadequate mobilization, impairment of blood supply, shearing stress in femoral neck fractures, interposition of muscles, and finally, infection. All these indicate the numerous factors to be recognized before definitive opinion can be stated.

Where the roentgenologist and orthopedic surgeon are in constant consultation, this book will be merely an aid to the radiologist. When the radiologist is in constant liaison with general practitioners, this book will improve his fundamental understanding of routine procedures. To residents in the field, it should prove valuable.

The reviewer does feel that this book should have mentioned, if only briefly, the overtreatment of fractures in the following conditions: fractures of the transverse processes of the lumbar vertebrae, fractures of the pelvic bones, undisplaced fractures of the patella, fractures of the distal fibula involving the ankle joint, and fractures of the head and neck of the humerus.

Stephen N. Tager, M.D.

Annual Review of Medicine. Volume 7. 1956. David A. Rytand, Editor. Cloth. Price, $7.00. Pp. 611, with illustrations and tables. Annual Reviews, Inc., Stanford, California, 1956.

This is an excellent review of selected subjects. The book is not intended to review the entire field of internal medicine in any one year, as the name might imply. Rather, the reviews, by well chosen authors, are limited to topics which vary from year to year. It should be noted that the present volume contains a

section entitled "Radioactivity Injury and Recovery from Radiation Exposure," by J. W. Howland, and one entitled "Hematopoietic Responses to Radiation Injury," by L. Jacobson.

The reviews are comprehensive. They not only deal with the recent literature but frequently refer to earlier papers. The bibliography that accompanies each subject is extensive.

A very worthwhile section of this work is the annotated list of reviews in medicine. This is a list of review articles on various subjects related to internal medicine which have appeared in the literature during the period from the previous list to December, 1955. Whereas this section might be of more value and more easily used with a different system of classifying the reviews, the authors note that improvements on classification will be forthcoming in later volumes.

Ronald C. Bishop, M.D.

Lungenkrebs und Bronchographie. Von Doz. Dr. H. Anacker, Giessen. Cloth. Price, $6.80. Pp. 78, with 45 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1955.

This is a rather short monograph on the bronchographic findings in patients with bronchogenic carcinoma. It is based on a study of more than 500 cases with bronchogenic carcinoma. Some detailed anatomical studies of 91 surgical specimens are reported. A positive roentgen diagnosis was possible in 82 per cent of the cases in which bronchography was performed. Positive bronchographic findings were present in almost all cases of bronchogenic carcinoma near the hilum, whereas the majority of the negative findings were found in those cases in which the lesion originated in the periphery. The bronchographic findings and their differential diagnoses are discussed in detail.

Of particular interest are the deductions which are drawn from the bronchography in regard to the origin of bronchogenic carcinoma. For the majority of the cases, the mouth of a segmental bronchus is thought to be the site of origin for the tumor. Anatomic and physiologic reasons for the prevalence of this site are discussed. The study is of considerable interest.

Richard Schatzki, M.D.

The Treatment of Fractures. Volume I. By Lorenz Böhler, M.D., Director of the Accident Hospital, Vienna; Professor of Accident Surgery, University of Vienna. Translated from the Thirteenth German Edition by Hans Tretter and others. Cloth. Price, $24.50. Pp. 1,104, with 1,721 illustrations. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16, New York, 1956.

The author meticulously presents the methods of treatment of injuries currently employed by himself and associates. He bases his recommendations upon 660,000 injury cases treated at the Accident Hospital in Vienna, in addition to his experiences in both World Wars and in Spain in 1937.

An organized plan of treatment is presented in detail for each specific injury, including his method of organization of a service for the treatment of traumatic injuries. This first volume is devoted to principles of the general treatment of injuries and the specific treatment of injuries of the skull, spinal column, pelvis, chest and abdomen, the shoulder girdle and upper extremity. The author describes his approach to gunshot wounds of the described areas. This survey of trauma includes a description of the factors the author believes predispose to failure in treatment in his case analyses. In each instance the specific materials to be used and the manner of their utilization are discussed in minute detail.

Many American and British surgeons will not agree with the author's method in every instance. They will, however, be stimulated by his methods, which are provocatively, rather than pedantically, presented. The volume is easy to read, well illustrated, and carefully indexed.

The material in this volume represents a fantastically large amount of work by the author and his associates. The volume justly deserves a place in the library of any surgeon interested in the treatment of trauma. It approaches the status of an encyclopedia in its completeness.

Frank P. Walsh, M.D.

BOOKS RECEIVED

Some Effects of Ionizing Radiation on Human Beings. Edited by E. P. Cronkite, V. P. Bond, and C. L. Dunham. From the Naval Medical Research Institute, Bethesda, Maryland; U. S. Naval Radiological Defense Laboratory, San Francisco, California; and Medical Department, Brookhaven

National Laboratory, Upton, New York. Paper Pp. 106, with illustrations and tables. United States Atomic Energy Commission, Oak Ridge, Tennessee. For sale by the Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C., 1956.

RADIOLOGY OF THE ALIMENTARY TRACT IN INFANCY. By Roy Astley, M.B., M.R.C.S., D.M.R., Radiologist, The Children's Hospital, Birmingham (United Birmingham Hospitals); Clinical Lecturer in Radiology and Lecturer in Radiological Anatomy, University of Birmingham. Cloth. $10.00. Pp. 287, with numerous figures. Edward Arnold (Publishers) Ltd., London. In United States: The Williams & Wilkins Co., Mount Royal and Guilford Avenues, Baltimore 2, Maryland.

ATLAS OF TUMORS OF THE NERVOUS SYSTEM. By H. M. Zimmerman, M.D., Chief, Laboratory Division, Montefiore Hospital, and Professor of Pathology, College of Physicians and Surgeons, Columbia University, New York City; Martin G. Netsky, M.D., Associate Neuropathologist and Associate Attending Physician in Neuropsychiatry, Montefiore Hospital, New York City. Presently Professor of Neuropathology and Associate Professor of Neurology, The Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, North Carolina; and Leo M. Davidoff, M.D., Attending Neurological Surgeon, Montefiore Hospital, and Professor and Chairman, Department of Surgery, Albert Einstein College of Medicine, New York City. Cloth. Price, $25.00. Pp. 191, with 277 illustrations. Lea & Febiger, Washington Square, Philadelphia 6, Pennsylvania, 1956.

Vol. 77, No. 2

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Genitourinary System

Balestra, G., and Delpino, B. In tema di rene a spugna e di nefrocalcinosi. (Concerning the sponge kidney and nephrocalcinosis.) *Radiol. med.*, Aug., 1956, *42*, 745–764. (From: Istituto di Radiologia dell'Università di Genova and Ospedale Civile di Genova-Sampierdarena, Genova, Italy.)

Lenarduzzi (*Radiol. med.*, 1939, *26*, 346) gave the name of sponge kidney to the roentgenologic appearance of a cystic dysplasia localized in the pyramids of the kidney. The microcavities may or may not contain calcifications.

Nephrocalcinosis is characterized by the presence in the renal parenchyma of diffuse, small calcifications, and the term is applied only to the case in which the calcifications are seen roentgenologically. The most frequent primary disorders responsible for the development of nephrocalcinosis are primary hyperparathyroidism, hyperchloremic acidosis and chronic pyelonephritis.

Neither the sponge kidney nor nephrocalcinosis presents any characteristic symptoms and their recognition depends only on the roentgenographic demonstration of microcavities in the former and calcifications in the latter.

The sponge kidney may be present only on one side, while in nephrocalcinosis both kidneys are affected.

The presence of small, scattered calcific deposits in one kidney as demonstrated in the scout film of the abdomen, and the filling of small parenchymal cavities following the intravenous injection of the contrast medium are findings more probative for a sponge kidney than for nephrocalcinosis.

Nevertheless one cannot exclude on the basis of the pathologic changes, the possibility that the findings described above may be present in nephrocalcinosis, where secondary cysts and/or tubular ectasias, with a few scattered calcifications, have been described.

The authors are of the opinion that the term sponge kidney should be adopted only in those cases which have positive findings on intravenous urography, when the changes are localized in one kidney alone. In all other cases the term nephrocalcinosis should be used without any consideration of the primary cause responsible for the changes.—*A. Govoni, M.D.*

Ottoman, Richard E., Woodruff, John H., Jr., Wilk, Stefan, and Isaac, Frank. The roentgen aspects of necrotizing renal papillitis. *Radiology*, Aug., 1956, *67*, 157–167. (Address: R. E. Ottoman, University of California Medical Center, Los Angeles 24, Calif.)

Diabetes mellitus and acute pyelonephritis are the two conditions in which acute necrosis of the renal papillae occurs most frequently as a complication. Less frequently it is observed in obstructive uropathy with superimposed infection. The onset of this complication should be suspected in any patient with these conditions whose clinical status suddenly becomes worse and in whom there is a decrease in urinary function and a rapid rise in the blood urea nitrogen.

Roentgenographically the findings observed in the 8 cases reported by the authors are as follows:

1. Calyceal haziness or unsharpness
2. Ulcerative or erosive papillary changes
3. Sinus formation
   (a) Intramedullary
   (b) Intrapapillary (ring shadows)
4. Parenchymal cavitation with communicating sinuses
5. Amputated calyces
6. Extruded papilla with secondary
   (a) Club-like papillary fossa
   (b) Ureteral or calyceal obstruction
   (c) Intrapelvic filling defect
7. Narrow infundibula, occasionally showing a "moth-eaten" margin
8. Decreased urinary function as seen on the intravenous pyelogram
9. Enlargement of the renal silhouette

The diagnosis of this complication is best made by retrograde pyelography. Histologic examination of bits of sloughed papilla passed in the urine or found in the bladder during cystoscopy will also give the diagnosis. The differential diagnosis includes pyelocanalicular backflow, renal tuberculosis, acute pyelonephritis without necrosis, radiolucent urinary calculi and neoplasms. While necrotizing renal papillitis is not a common entity, its early and more frequent recognition may permit more prompt institution of appropriate therapy with greater prolongation of life.—*Donald N. Dysart, M.D.*

Schlungbaum, W., and Billion, H. Untersuchungen der Verteilung von radioaktivem Urografin im menschlichen Organismus. (Investigation concerning the distribution of radioactive urografin in the human organism.) *Klin. Wchnschr.*, June, 1956, *34*, 633–635. (From: Strahleninstitut der Freien Universität Berlin am Städtischen Krankenhaus Westend, Berlin, Germany.)

For tracer studies radioiodine was added to urografin (renografin) in a series of 10 patients of various ages. Six received 400 mg. and the remaining 4, a dose of 2,400 mg. of urografin intravenously. The blood was tested five, fifteen, thirty, forty-five and

sixty minutes after the injection, and in 4 cases activity measurements of the feces were made after twenty-four and forty-eight hours. The activity of the individual dose was approximately 20μ/c.

The concentration of urografin in the serum showed a biphasic course. In the beginning the half-life time was rapid, amounting to six minutes. The second phase showed a half-life time of one hundred and fifteen minutes. When 2,400 mg. of urografin was administered, instead of 400 mg., the serum level of urografin did not become increased.

In the urine 60 per cent was excreted in three hours and 90 per cent in twenty-four hours. In the fecal material an excretion of only one to two per cent was discovered after forty-eight hours.

Comparison with radioactive perabrodil M revealed the same serum values in the first phase, but in the second phase the serum values were prolonged with radioactive urografin. A distinct difference was noted in the excretion time. While perabrodil was excreted by the kidneys up to 90 per cent within three hours, the corresponding values for urografin were barely 60 per cent.

It is concluded that for practical clearance determinations urografin is less suitable than perabrodil M because of delayed excretion.—*Ernest Kraft, M.D.*

FINBY, NATHANIEL, POKER, NATHAN, and EVANS, JOHN A. Ninety per cent hypaque for rapid intravenous roentgenography; preliminary report. *Radiology*, Aug., 1956, *67*, 244–246. (Address: N. Finby, New York Hospital, 525 East 68th St., New York 21, N. Y.)

Highly concentrated iodinated contrast media are needed for rapid intravenous injection in angiocardiography and nephrotomography. The present study documents the use of 90 per cent hypaque in 50 consecutive patients, 22 of whom received 2 injections, usually within a fifteen minute interval. Of the patients examined, 31 were males and 19 females, the youngest being thirteen and the oldest seventy-six years of age. The amount of medium used per injection varied between 30 and 50 cc.

The rapid injection of concentrated contrast material for angiocardiography or nephrotomography is frequently associated with symptoms. The authors have therefore, contributed important information in their report of a comparison of the reactions noted following injections of 90 per cent hypaque with those seen following the use of 70 per cent urokon.

It is noteworthy that a significant decrease in nausea, cough, arm pain, vomiting, weakness, urticaria, and thirst was observed in those patients who received 90 per cent hypaque. Vertigo occurred in 14 per cent and flush was noted in 90 per cent of the patients. A metallic or acid taste was observed in 5 instances, and once a transient tightness in the chest was noted. Eight of 10 patients receiving a single injection of 90 per cent hypaque after transcutaneous insertion of the Robb-Steinberg needle had thrombosis at the site of injection. Only one instance of significant phlebitis occurred in the entire group of 50 patients. This complication responded quickly to routine measures.

Opacification of the cardiovascular and pulmonary structures in angiocardiography and of the aorta and kidneys in nephrotomography with 90 per cent hypaque was found to be comparable to that obtained with 70 per cent urokon.—*A. R. Watson, M.D.*

COUVELAIRE, R., and AUVERT, J. (Paris, France.) La phlébographie cave inférieure dans l'exploration des tumeurs du rein droit. (Phlebography of the inferior vena cava in the exploration of right kidney tumors.) *J. d'urol.*, Jan.–Feb., 1956, *62*, 21–40.

Special examinations of the kidneys like excretory urography, retrograde pyelography and aortography fail to demonstrate the renal veins. In cases of right kidney tumors the authors show that venographic studies give valuable information to the surgeon preoperatively.

The technical approach is described in the following manner: A plastic catheter with a caliber of 3 to 4 mm. is inserted into the right internal saphenous vein below Poupart's ligament for a distance of 20 to 25 cm. When the tip reaches the bifurcation of the inferior vena cava, the tilt table is inclined 20 degrees to retard the venous flow. The patient is then rotated into the right oblique position so that the vena cava can be projected outside of the spinal column and possible pressure from a right kidney tumor will be lessened. At first, 5 to 10 cc. of a 70 per cent iodine contrast solution is injected and a trial exposure is made. This is followed by a rapid injection of 40 cc. within a period of three to four seconds. The first exposure is still made during the injection and is followed by additional exposures in rapid succession. An excretory urogram is obtained ten minutes later.

The right kidney is closer to the inferior vena cava than the left and the right renal vein is shorter than the left. Therefore, phlebograms are of real value only in tumors of the right kidney. Since renal veins are frequently invaded by tumor thrombi regardless of the size of the tumor, phlebography is recommended not only in large tumors, but also when the neoplasm is small.

Normally, the opacified vena cava becomes slightly narrowed at the level of the renal veins due to influx of nonopaque blood. The renal veins are usually bypassed and, therefore, not visualized. The authors, however, have blocked the vena cava higher up in dogs with a balloon catheter and, thereby, have succeeded in filling the renal veins and branches with contrast material.

Abnormal phlebograms show extrinsic compression of the vena cava so that the opaque column be-

comes narrowed and encroached upon and curves toward the left of the midline. There may also be backflow into the left common iliac vein which is not observed in normal cases. The contour of the vena cava remains smooth and regular. When the renal vein and its orifice are the site of a thrombus, however, a typical indentation with an irregular contour can be observed in the opaque column. In cases with a floating tumor thrombus a central filling defect is sometimes noted in the vena cava. In far advanced cases of tumor extension total block of the venous flow is experienced. This is associated with marked backflow into the iliac veins and with collateral circulation. Attention is also called to drainage through collateral perirenal veins when the renal veins are blocked.

Seven representative cases are reported and illustrated with reproductions of 14 roentgenograms.—*Ernest Kraft, M.D.*

Skeletal System

Paltrinieri, G. (Brescia, Italy.) Le syndrome de Morgagni et ses rapports avec l'intersexualité. (Morgagni's syndrome and its relationship to intersexuality.) *J. de radiol. et d'électrol. & Arch. d'électric. méd.*, March–April, 1956, *37*, 145–157.

History and review of this syndrome is presented with appropriate quotations from J. B. Morgagni's original publication (1761). The contributions of authors such as Morel, Moore, Greig, Pende, Stewart and others, to the elucidation of the syndrome and its variations are discussed.

The main anatomic finding is a patchy, and at times, diffuse hyperostosis of the inner table of the frontal bone. Occasionally localized hyperostosis can be noted at the base of the cranium. It occurs predominantly in women of about forty years of age. Neuropsychic disturbances of mild to severe degree and obesity are often associated with the syndrome, although some cases may be underweight. Certain relationships to menopause have led a few authors to think of it as a mild degree of Cushing's syndrome.

Three developmental phases have been described by Razo, i.e., osteoblastic, osteoclastic, and finally a certain degree of bony involution of the thickened inner table and of the membranous portion of the frontal bone. The meningeal involvement and thickening appear to be secondary. In case of such thickening within the anterior part of the base of the cranium, infection should be suspected as the causative factor because of the contiguity of the paranasal sinuses.

Out of 1,000 cases of skull roentgenograms, the author found 18 instances showing hyperostosis of the inner table of the frontal bone and 6 cases of membranous hyperostosis, one of which was frontoparietal as to site. Out of these 24 cases, 23 were women. The only male case was a fifty-seven year old patient exhibiting a pituitary type of gigantism with many feminine traits and physical findings of pseudohyperostosis of the inner table of the frontal bone.

The author also describes in detail the clinical, laboratory and roentgen findings of a fourteen year old girl showing hyperostosis of the inner table of the skull.

No clinical laboratory or roentgenographic findings could link Morgagni's syndrome with pituitary dysfunction, according to the author. Thus the obesity and virilism and the neuropsychic symptoms in Morgagni's syndrome do not correspond to those due to pituitary, suprarenal, ovarian or other endocrine disorders. Intersexuality, i.e., virilism and hirsutism in the women patients, and feminine traits in the single male patient are stressed in pointing out sex linked (genotypic) heredity as the etiologic basis of this syndrome. Other hyperostotic syndromes can be traced to circulatory (inflammatory or allergic meningeal reaction), endocrine or metabolic disturbances. Hence, the author feels justified to suggest for Morgagni's syndrome, the more descriptive name of "Disease of Morgagni—intersexuality or hyperostosis-intersexuality."—*Jirair N. Sarian, M.D.*

Singleton, Edward B., Thomas, John R., Worthington, William W., and Hild, John R. Progressive diaphyseal dysplasia (Engelmann's Disease). *Radiology*, Aug., 1956, *67*, 233–241. (From: Departments of Radiology and Pediatrics, Texas Children's Hospital, Houston, Texas and the Norsworthy Pathology Research Laboratory, Hermann Hospital, Houston, Texas.)

Engelmann's disease (progressive diaphyseal dysplasia) is a rare disorder of bone characterized by weakness and underdevelopment of the muscles of the extremities and sclerosis and fusiform enlargement of the diaphyses of the long bones. It was first described in 1929 by Engelmann who referred to it as "osteopathia hyperostotica sclerotisans multiplex infantilis."

The previously reported cases (16) are reviewed and one additional case is added. This patient, a white male, aged five and one half years, developed a peculiar gait at the age of two years, complained of ankle pain and leg cramps and tired easily. Roentgenographic studies of the skeleton showed symmetrical fusiform sclerosis involving the diaphysis of each tibia, fibula, femur, humerus, ulna and radius. The cortex of the affected bones was thickened and the medullary cavity was narrowed. Large localized areas of rarefaction, possibly around nutrient vessels, were present in the mid-portion of each tibia and slightly higher than this in each femur. The outstanding pathologic features of the biopsy tissues were muscle atrophy and sclerosis of bone associated with thickening of the walls and reduction in size of

the lumens of the arterioles of the periosteum, bone marrow, and subcutaneous tissue.

Etiology of this disease is unknown. Hereditary factors have been conspicuously absent in most of the reported cases. Serologic studies have been consistently negative, and laboratory studies have provided no clue as to the cause.—*A. W. Sommer, M.D.*

PONSETI, IGNACIO V. Legg-Perthes disease; observations on pathological changes in two cases. *J. Bone & Joint Surg.*, July, 1956, *38-A*, 739–750. (From: Department of Orthopaedic Surgery, State University of Iowa, Iowa City, Iowa.)

It appears that the primary lesion in Legg-Perthes disease is faulty epiphyseal-plate cartilage and that necrosis of the head follows when the blood supply is interrupted. This is similar to the lesions noted in rats on a special diet high in aminonitriles. Initially the ground substance becomes defective with fibrillation and rent formation and the cartilage cells react by proliferation and cluster.

It has been found that in children with Legg-Perthes disease other ossification centers may be deranged, but the peculiar blood supply of the femoral head may account for the more serious consequences here.

In the younger age group the epiphyseal line is nearly horizontal and the periosteum thick and strong; the retinacular vessels must penetrate thick cartilage to reach the center of ossification of the head and the artery of the ligamentum teres does not reach the ossified portion of the head till twelve to fourteen years. All these factors apparently tend toward development of Legg-Perthes disease when there is a faulty epiphyseal plate, while in the older age group with a faulty epiphyseal plate, epiphysiolysis is more apt to develop, since the epiphyseal plate is more nearly vertical, the periosteum thinner and the retinacular vessels reach it after piercing mature joint cartilage.

Biopsies in 2 children with early Legg-Perthes disease showed a necrotic femoral head and a disrupted epiphyseal plate. The epiphyseal plate cartilage was fibrillated and cracked, the cells clustered and the line of endochondral ossification irregular. Tongues of deranged cartilage and cartilaginous islands were noted deep in the metaphysis and femoral head. There was some fibrillation in areas of the joint cartilage adjacent to the subchondral plate. There was no osteoid tissue seen in the metaphysis. The bone marrow was normal. No inflammatory cells were shown.

The microscopic changes in Legg-Perthes disease are similar to those shown in patients with slipping of the upper femoral epiphysis and to the epiphyseal plate lesions observed in young rats fed diets high in aminonitriles. Adult rats on this diet for a few months developed joint lesions similar to osteoarthritis in humans.—*Martha Mottram, M.D.*

ROSS, F. G. M. Radiosensitive bone tumours—radiological aspects. *J. Fac. Radiologists*, April, 1956, *7*, 242–252. (From: United Bristol Hospitals, Bristol, England.)

This paper is based on a study of the radiosensitive bone tumors encountered in a group of 567 bone tumors coming under study at the Bristol Bone Tumour Register. Hewer (1956) has recently resolved all the cases previously classified by the Bone Tumour Register as Ewing's tumor into either reticulosarcoma or secondary neuroblastoma. The paper is confined to a description of the roentgenologic appearances of reticulosarcoma of bone of which there were 14 in the Register, and a comparison between it and secondary neuroblastoma. Malignant synovioma is considered briefly as a separate entity.

Ross groups the cases of reticulosarcoma as: (a) single primary focus in bone, 7 cases; (b) multiple primary foci in bone, 2 cases; and (c) primary soft tissue reticulosarcoma secondarily involving bone, 5 cases.

Reticulosarcoma almost always arises in the medulla, being most common in the long bones. It may originate in the epiphysis, but more often it may begin in or away from the center of the diaphysis. The major direction of growth is along the shaft, and the lesion may or may not cross the epiphyseal line. Early, the tumor appears within the medulla as a group of small localized translucencies, with ill-defined margins. As the lesion spreads up and down the shaft the smaller osteolytic areas become confluent. As further growth occurs the cortex is destroyed in a patchy manner similar to that seen in the medulla and there is rapid loss of the corticomedullary junction. Layered or amorphous periosteal new bone, or fine bony spiculation may be seen after extension of the lesion through the cortex. A small periosteal reactive triangle may be seen at the periphery of the lesion. Medullary bone production may occur, but bone destruction is always present and almost always predominant. A soft tissue tumor, usually small and ill-defined, is occasionally seen. Response to irradiation is rapid and dramatic. Bony re-organization occurs, and the final result is an area in which there is slightly coarse irregular trabeculation with some general increase in density and occasionally small denser areas and some cortical thickening ("scar bone"). Only one of the primary cases in the Bone Tumour Register showed later evidence of distant bone metastasis.

Secondary neuroblastoma involves femur, humerus, pelvis, skull, and tibia in that order. Primary intrathoracic neuroblastomas are paravertebral in origin. Metastatic rib lesions are usually more laterally situated and rib destruction is often extensive. In secondary neuroblastoma the lesions are usually extensive when first seen and may involve the shaft of a long bone from one epiphysis to the other. There is multiple bone involvement in up to 70 per cent of cases and this, plus the fact that the lesions tend to

be bilateral, is helpful in differential diagnosis. Early, the sites of predilection are the medial aspects of the distal femoral and proximal humeral metaphyses. Medullary bone destruction is always present, with the same appearance as in reticulosarcoma, except that it eventually involves a much greater length of shaft. Cortical destruction is common, as also is layered periosteal new bone formation. Spiculated new bone formation extending out into the usually present soft tissue tumor is a conspicuous feature. Medullary bone production seen in about a third of cases is most frequent in the pelvis. The soft tissue tumor may be large, has well-defined margins and may extend from one or all surfaces of the affected bone. The well-known changes in the bones of the vault of the skull are seen occasionally, and are the same as the bone lesions seen elsewhere, with diastasis of sutures produced by increased intracranial pressure. All these bone changes may develop very rapidly within a few weeks to a month or more. The response to radiation is initially good, but less rapid than with reticulosarcoma. Bone repair may be marked, but temporary and the bone rarely returns to normal. The soft tissue tumor decreases in size and there is re-ossification of the lesion and organization of periosteal new bone formation, with abnormally wide and dense appearance of the bone. Eventual further destruction at the original site is inevitable.

Malignant synovioma usually arises in proximity to a joint, but may arise in relation to tendon sheaths and bursae anywhere in the body. These are extraosseous in origin and rarely invade the bone by direct extension from the primary tumor or by distant metastasis. Pulmonary metastases are more common than metastases to bone. Malignant synoviomata are most common in relation to the knee joint and next most common in the arm. On roentgen examination a well-defined homogeneous soft tissue tumor with either a regular or lobulated outline is seen. It varies in size from about 2 to 16 cm. in diameter. Irregular areas of calcification may be seen within the soft tissue tumor, either before or after it has been interfered with by surgery or roentgen therapy. The tumor grows slowly. There is apt to be irregular destruction of cortex and underlying medulla. Destruction of bone is usually predominant but some bone sclerosis may occur. Disuse osteoporosis may be seen, especially distal to the tumor, if function of the limb is impaired. With roentgen therapy, the soft tissue tumor may decrease considerably in size but the author did not find any in which the tumor completely disappeared nor in which the destroyed bone had regenerated.—*Samuel G. Henderson, M.D.*

Kunkel, Melvin G., Dahlin, David C., and Young, H. Herman. Benign chondroblastoma. *J. Bone & Joint Surg.*, July, 1956, *38-A*, 817–826. (From: Section of Orthopaedic Surgery, and Section of Surgical Pathology, Mayo Clinic and Mayo Foundation, Rochester, Minn.)

Benign chondroblastoma has been classified as a variant of giant-cell tumor and by Codman as epiphyseal chondromatous giant-cell tumor. There has been a difference of opinion regarding malignancy in some cases, but Lichtenstein, who designated it as a distinct entity, denies that it is ever malignant.

This report concerns 16 benign chondroblastomas found at the Mayo Clinic, less than 1 per cent of their primary bone tumors. The ages ranged from eight to fifty-four, the majority being between fourteen and twenty-one years. There were 10 males and 6 females. Ten of the 16 tumors were in the tubular bones and all but one of these was entirely or partially epiphyseal. The location of the other 6 tumors was such that they could have been associated with secondary centers of ossification.

The presenting complaint was pain in the majority of instances with instability, limitation of motion, muscular wasting, stiffness, weakness, and limping. The symptoms averaged two years in duration before diagnosis. A mass was palpable in 7 cases. There were no pathological fractures.

Roentgen examination showed a round or oval fuzzy mottled, rarefied lesion in the epiphysis or extending from it into the adjacent bone. When the metaphysis is involved the overlying cortex may be expanded or destroyed. Some lesions showed a well-defined line of marginal sclerotic bone. Those in the flat bones were variable in appearance and 2 suggested malignancy. The tumors ranged in size from 1 cm. to one which destroyed most of the scapula.

Grossly the lesions were dark blue-gray, either friable or rubbery. Some showed cystic, necrotic or hemorrhagic foci. The predominant cell was the chondroblast. Some of these cells showed mitotic figures. There was little interstitial substance. There were multiple nucleated giant cells. Chondroid material and occasionally hyaline cartilage occurred in varying amounts, and in older portions sometimes calcified (although this was not noted as a roentgen feature and is presumably microscopic). They appeared closely related to chondromyxoid fibroma of bone. One of Kunkel's patients had a chondroblastoma which recurred nine years later as a chondromyxoid fibroma.

It is necessary to do a biopsy in order to establish the diagnosis. The lesion most resembling it is giant cell tumor. The authors' series had a variety of treatments including amputation, biopsy alone, curettage, resection and roentgen therapy. Codman recommends roentgen therapy only.

Since these lesions are benign they should have conservative treatment. The recurrence in 1 case and persistence in another indicates that thorough removal is necessary. Kunkel recommends that irradiation should not be used because of possible radiation induced malignancy, unless anatomically, the lesion

cannot be excised or resected.—*Martha Mottram, M.D.*

DAVIES, A. G. M. Bone changes in Kaposi's sarcoma; an analysis of 15 cases occurring in Bantu Africans. *J. Fac. Radiologists*, July, 1956, *8*, 32–40. (From: Mulago Hospital, Kampala, Uganda, Africa.)

Idiopathic multiple hemorrhagic sarcoma, originally described by Kaposi in 1872, is a disease of unknown etiology, characterized by the appearance in the skin of multiple nodules and placques. These nodules and placques are usually found on the extremities, more often on the legs. The nodules vary in size from the head of a pin to a cherry or larger. While the early lesions are usually superficial, in the later stages the nodules may infiltrate deeply and produce hard swelling of the hands, feet, arms and legs. Histologically, the cardinal changes occur in the dermis and consist of new capillary formation, mainly made up of blood vessels with a few lymph vessels and connective tissue hyperplasia. Satenstein recognized 3 pathologic processes—inflammatory, granulomatous and neoplastic. The picture in the neoplastic type varies according to the elements involved. It may resemble an angioma or lymphangioma, or a mixture of both, a fibroma, angiofibroma, angiosarcoma, or a spindle-cell sarcoma. The disease is relatively common among the Eastern Europeans but was first reported in a full-blooded Negro in 1931. The present paper deals with 15 of 33 patients from the records of the Cancer Registry of the Medical School of Makerere College occurring from January 1953 to May 1955, roentgen examination having been made in these 15 cases. In all the cases, the dominant lesion was the nodule. The diagnosis was proved in every case by a nodule biopsy, which showed the typical histologic appearance.

Following are some of the roentgenologic changes: Uniform rarefaction was found in bones of the feet in 10 cases and in 6 of the 33 lower-limb long bones examined. These showed expansion of the medullary cavity with thinning of cortex from within. Localized areas of intense rarefaction were ill-defined and irregular in shape, with scanty, partially "rubbed-out" trabeculae, most commonly found in the spongiosa of the bases and heads of the phalanges of the feet. Cyst-like areas, varying in size from 2 to 10 mm. in diameter, had clear-cut borders and round or oval shapes, with no evidence of a cyst wall and no surrounding bone or periosteal reaction. The clear cyst-like spaces may roentgenologically resemble the pseudocysts seen in the extremities in sarcoidosis. Superficial cortical erosions were present in the bones of 10 of the 15 cases examined, being most common in the phalanges and metatarsals of the feet. The erosion may be ragged, suggesting malignant infiltration or it may be well-defined and smooth as from pressure erosion. Complete destruction of bone found in 1 case resembled a purely osteolytic bone sarcoma. Calcification of about 4 cm. of the first plantar metatarsal artery of the left foot was found in 1 case.

In differential diagnosis one must consider: multiple myeloma and osteolytic metastases (lesions of Kaposi, mainly confined to the bones of the extremities), gout (rarely seen in Africans and primarily affecting the articular surfaces of the joints), multiple chondromata (clear zones produced by chondromata are surrounded by a line of increased density), sarcoidosis (neither cortical erosion nor generalized rarefaction is a feature of sarcoidosis), and Madura foot (the osteolytic involvement in Madura foot is less well-defined, has ragged margins, mainly attacks first the bases of the metatarsals, and is usually accompanied by bone production).—*Samuel G. Henderson, M.D.*

SELBY, HENRY M., SHERMAN, ROBERT S., and PACK, GEORGE T. A roentgen study of bone metastases from melanoma. *Radiology*, Aug., 1956, *67*, 224–228. (Address: H. M. Selby, Memorial Cancer Center, 444 E. 68th St., New York 21, N. Y.)

This report discusses the roentgenographic manifestations of bone metastases from melanoma and is based on a study of 33 patients with histologically proven melanoma. Seventeen patients had solitary metastases located as follows: ribs, 6; skull, 4; pelvis, 2; spine, 2; humerus, 1; tibia, 1; and paranasal sinuses 1.

Where multiple metastases were present, up to 12 bones were involved and in some there were many involved areas in one bone. Of the 33 cases, the long bones were involved in 6, the flat bones in 27, and the spine in 9. The location in the long bones was equally in the diaphyseal and metaphyseal regions.

No specific roentgen appearance could be found. Most of the lesions were osteolytic (29 patients) while a few (4 patients) were osteoblastic to varying degrees. They were usually oval in shape, medullary in location, and showed cortical destruction. A soft tissue component was noted in 16 patients.

Pathological fractures occurred in 15 patients, with the ribs involved in 7. Size of the lesions varied from 5 mm. to 18 cm. in diameter with an average of about 3 cm.—*Donald N. Dysart, M.D.*

ORLANDINI, I., BASSANI, G., and CASACCI, A. La formazione e l'evoluzione del callo osseo di riparazione delle fratture. (The formation and evolution of bone callus in the repair of fractures.) *Ann. radiol. diag.*, 1956, *29*, 254–301. (From: Istituto di Radiologia Medica and Università di Parma, Istituto di Clinica Ortopedica, Parma, Italy.)

The animals used in this experiment were rabbits.

The bones of the forelimb were fractured and the animals were sacrificed at intervals of 4, 8, 12, 16, 20, 24, 31, 38, 45, 60 and 90 days after the fracture was inflicted. Studies of these bones using microradiography were carried out which permitted accurate analysis of the intimate bone structure. The evolution and formation of bone callus could be accurately recorded and studies.

The authors remind us that these studies were conducted on animals of rather small size and young in age. Many of the theories for callus formation are discussed. The article contains numerous microradiographs.—*Peter E. Russo, M.D.*

Blood and Lymph System

Amundsen, A. K., Amundsen, P., and Müller, O. Blood pressure and heart rate during angiocardiography, abdominal aortography, and arteriography of the lower extremities. *Acta radiol.*, June, 1956, *45*, 452–458. (From: Roentgen Department, Anaesthetic Department, and University Institute for Respiratory Physiology, Ullevål Sykehus, Oslo, Norway.)

The authors measured the blood pressure in the aorta at frequent intervals during angiocardiography, aortography and peripheral arteriography using nycotrast (diodone) 70 per cent or 50 per cent. They found that in angiocardiography the blood pressure and pulse usually remained constant during and immediately following the injection. By ten seconds there was sometimes a fall in aortic blood pressure and by twenty seconds there was always a definite decrease in pressure. This continued for thirty and forty seconds after the injection with a mean of 30 and 40 mm. Hg respectively. One minute after injection the pressure started to rise again in all cases.

The heart rate decreased slightly within the first ten seconds and there was usually a slight tachycardia afterward, although 3 cases developed a marked bradycardia and one developed a sino-auricular block.

In aortography a slight increase in blood pressure occurred during injection and this was followed by a marked fall within ten seconds. This reached a maximum mean fall of 39 mm. Hg in forty seconds. By sixty seconds the pressure had nearly always started to rise, but only 1 out of 6 had reached its pre-injection level four minutes after the injection. As the pressure fell, the heart rate increased; it decreased as the blood pressure was restored. When saline was injected the pressure and heart rate reactions during the injection and for the following ten seconds were very similar to those recorded in ordinary aortographies, but by twenty seconds after the injection the effect had disappeared almost completely.

The reactions to peripheral arteriography were very similar to those in aortography but of less magnitude.

It is concluded that the blood pressure drops earlier during aortography than during angiocardiography due to a peripheral vasodilatation effect of the contrast medium. During aortography the heart rate varies as if dependent on the aortic-carotid sinus reflex, but during angiocardiography the early slowing of the heart rate is probably the result of vagal stimulation or the action of the contrast medium directly on the heart muscle.—*Arthur E. Childe, M.D.*

Celis, Alejandro, Cicero, Raúl, del Castillo, Hermilo, and Arce G., Enrique. Temporary arrest of the contrast medium in angiocardiography. *Acta radiol.*, May, 1956, *45*, 341–351). (From: Department of Chest Diseases, (Pab. 27), Hospital General, México D. F.)

Angiocardiographic studies were done on 58 patients who had normal cardiovascular systems while performing the Valsalva's maneuver. Temporary arrest of circulation was achieved with increased roentgenographic opacity of the structure studied.

With the tip of the catheter inserted through the external jugular vein, it was found that the caliber of the superior vena cava was reduced, and in some cases, the azygos and left innominate veins were visualized. There was undulation of the margins of the superior vena cava and frequently there was marked narrowing of its terminal portion.

At times, the superior vena cava and the right atrium may be demonstrated, while at other times, only the atrium or the right ventricle visualize. The right atrium is reduced in size and has sharply defined margins. The auricular surface of the tricuspid valve becomes a clear cut straight line.

If the tip of the catheter is inserted into the inferior segment of the superior vena cava, the atrium becomes continuous with the terminal portion of the inferior vena cava including the superior hepatic veins. If the tip of the catheter is placed in the right auricle, a separate visualization of the atrium is obtained, and its walls are wavy in outline. When the tip of the catheter is inserted into the right ventricle, isolated visualization of the chamber may be obtained which will be reduced in size. If the pulmonary artery simultaneously visualizes, it will do so with less contrast.

Pulmonary circulation is altered so that the pulmonary artery is larger and the sigmoid valves close. Simultaneous representation of the arteries and veins in the lungs are not obtained under these circumstances.

When injection is made into the femoral vein, its advance is arrested somewhere in the inferior vena cava. The inferior vena cava shows undulation with filling of its tributaries.—*George A. Miller, M.D.*

SOLOFF, LOUIS A., and ZATUCHNI, JACOB. The angiocardiographic diagnosis of left atrial thrombosis. *Circulation*, July, 1956, *14*, 25–32. (From: Temple University School of Medcine and Hospital, Philadelphia, Pa.)

Five cases are described representing the first report of their visualization by angiocardiography. Thrombi in the left atrium had previously been diagnosed preoperatively only when they were sufficiently calcified to be demonstrated on conventional roentgenograms.

In mitral stenosis the left atrium is densely and homogeneously opacified by angiocardiography and this persists for a longer period than in the normal. Therefore, the left atrium is particularly suitable for angiocardiographic visualization of thrombi. The authors use the posteroanterior and the left lateral projections. The left lateral projection is better because the extreme posterior wall of the left atrium is visualized only in this projection and it is from this wall that a mural thrombus almost always arises.

The diagnosis of a mural thrombus within the left atrium by angiocardiography is based upon the finding of a filling defect and irregular margination of the posterior wall. A ball valve thrombus is suggested by the finding of a filling defect within the left atrium unattached to the atrial wall and small compared to a myxoma. Left auricular thrombosis is suggested by irregular margination of the junction of the auricle and the atrium and sparse or no visualization of the auricle.—*John F. Weigen, M.D.*

FIGLEY, MELVIN M., NORDENSTRÖM, BJÖRN, STERN, AARON M., and SLOAN, HERBERT. Angiocardiographic mixing defects as indicators of left to right shunts. *Acta radiol.*, June, 1956, *45*, 425–437. (From Departments of Radiology, Pediatrics, and Surgery, University of Michigan, Ann Arbor, Mich.)

Seventy-five patients with verified left to right shunts were used in the study.

Normally, areas of nonuniformity in concentration of contrast material may be seen due to incomplete mixing of the material and blood in venous angiocardiography. At times, mixing defects may indicate an underlying anatomic abnormality due to a congenital intracardiac or extracardiac left to right shunt. In cases with atrial or ventricular septal defects, this sign is not frequently seen. Mixing defects in the pulmonary artery are commonly observed with a patent ductus arteriosus and this sign can be considered almost specific.

A discussion of factors such as roentgenographic technique, projections, timing, volume of the shunt and right ventricular pressure is also presented.—*George A. Miller, M.D.*

RODRIGUEZ, HECTOR F., GARDNER, FRANK H., and DIAZ-BONNET, RAFAEL. Splenoportography: A valuable adjunct in the study of portal hypertension. *Am. J. M. Sc.*, July, 1956, *232*, 1–7. (From: Bayamon District Hospital, Bayamon, Puerto Rico.)

Percutaneous splenoportography is a relatively simple procedure, without serious complications, which is advantageous in defining the types and degrees of portal hypertension. Following intrasplenic injection of diodrast, a single anteroposterior roentgenogram of the abdomen is sufficient for diagnosis. This report concerns 28 splenoportographies in a variety of conditions.

In patients without portal hypertension, the opaque material promptly leaves the spleen with visualization of smooth, normal caliber splenic and portal veins and intrahepatic branches. No collateral branches are visualized. The opaque material clears the spleen in six to twelve hours. In portal hypertension the splenic vein is tortuous and both the splenic and portal veins are wider than normal. The intrahepatic radicles are incompletely filled and collateral veins often are visualized. Esophageal varices may be seen by this method even in cases where they are not easily demonstrated by the methods usually employed.

In extrahepatic obstruction of the portal vein there is dilatation of the proximal portion of the splenic vein but the distal portion of the splenic vein and the portal vein are not visualized. Large collaterals are often seen.

The procedure is useful in the postoperative evaluation of patency and efficacy of a portocaval shunt. Reduction in size of the preoperatively dilated splenic vein is indicative of decrease in portal hypertension.—*Michael J. Healy, M.D.*

ALBALA, MAURICE M., BARRICK, CLAUDE W., and JENKINSON, EDWARD L. Vertebral transskeletal phlebography. *Radiology*, Aug., 1956, *67*, 229–232. (Address: M. Albala, 700 North Michigan Ave., Chicago, Ill.)

A simple safe procedure for visualization of the thoracolumbar veins by trans-skeletal injection of diodrast into the spinous processes of the lower dorsal and lumbar vertebrae or lower ribs is described. After injection of the skin and periosteum with novocain, 20 cc. of 70 per cent diodrast is injected rapidly through a small marrow needle into the spinous process or rib and a single lateral exposure is made immediately upon the termination of the injection. When a lower thoracic spinous process was injected, a variable amount of diodrast was seen in the durorachidian vein system, as well as in several intercostal veins and the azygos vein up to its point of entrance into the superior vena cava. With injection of a lumbar spinous process, several lumbar intercostal veins and an unpredictable number of durorachidian veins were filled. When a lower rib was injected, the intercostal and azygos system was strikingly well shown

but the durorachidian veins were not filled. The inferior vena cava could be demonstrated at its origin when the fourth and fifth lumbar spinous processes were injected.

The vascular bed of the vertebral column has no valves and an unusually rich anastomosis with other veins and is subject to reversals of the blood flow with changes in the intra-abdominal and intrathoracic pressure. Theoretically, these vascular connections provide the explanation for an important mode of metastasis of breast and prostatic tumors, cerebral emboli following injection of air into the retroperitoneal space or pneumothorax, and the cerebral extension of bronchogenic tumors.

Vertebral phlebography provides a new means for the demonstration of mediastinal masses causing obstruction of the superior vena cava and azygos veins and may prove of value in the demonstration of retroperitoneal masses that impinge upon the inferior vena cava.—*Harold C. Hamilton, M.D.*

Levy, Laurence F. Granuloma of the neck following thorotrast angiography. *Brit. J. Surg.*, May, 1956, *43*, 638–641. (From: Department of Neurology and Neurosurgery, McGill University and the Montreal Neurological Institute, Montreal, Quebec.)

Although no malignant change or other disease can directly be traced to the effect of thorotrast, following its use there has been reported sarcoma of the liver, fibrosis of the liver, aplastic anemia, and generalized necrosis of the lymph nodes.

The author reports 2 cases in which thorotrast was injected extravascularly during cerebral angiography. Follow-up studies showed that the material remained in the region. A mass developed and on removal was found to be a granuloma. These are not thought to be premalignant, but may cause difficulty because of pressure on adjacent structures.

Conservative treatment is recommended.—*George L. Sackett, Jr., M.D.*

Weens, H. Stephen, and Marin, Carlos A. Infantile arteriosclerosis. *Radiology*, Aug., 1956, *67*, 168–174. (Address: H. S. Weens, Emory University, 69 Butler St., S. E., Atlanta, Ga.)

Calcification and sclerosis of the arteries in infants and young children may be encountered in a variety of clinical conditions. Foremost among these are disorders in which disturbances of the calcium and phosphorus metabolism play an important role. Thus infantile arteriosclerosis has been recognized in renal disease, hyperparathyroidism, and vitamin D intoxication. Similar arterial changes have been noted in progeria, in which the arteriosclerotic process is associated with extensive growth disturbances and profound metabolic changes. Aside from these conditions, infantile arteriosclerosis has been observed in an increasing number of cases in which, at present, no obvious cause is established.

The pathologic changes noted in infantile arteriosclerosis of unknown etiology consist of fragmentation of the internal elastic membrane, intimal and medial calcification, and fibroblastic proliferation of the intima. Arteries of medium size are predominantly affected. The similarity of the lesions described here to those occurring in derangements of calcium and phosphorus metabolism make an explanation for the arterial changes on a metabolic basis attractive. So far, clinical, pathologic, and laboratory observations have not afforded substantiation of such a concept. Possibly metabolic changes may occur which are so subtile that they have not been recognized. Recent pathologic and experimental studies have stressed the role of the elastic elements of the arteries in the development of arteriosclerosis. As histologic observations indicate that the incipient changes of infantile arteriosclerosis affect the internal elastic membrane, it is conceivable that a deficiency of elastic elements may, in itself or in association with extraneous factors, lead to the arterial changes. It is suggested that genetic factors may predispose to the development of the condition.

The authors report 2 cases which illustrate the pertinent clinical roentgenologic, and pathologic features of infantile arteriosclerosis of unknown etiology. Emphasis is placed upon roentgenologic examination as the most important means of establishing a diagnosis during life.—*A. R. Watson, M.D.*

Bruun, Sven, and Engeset, Arnfinn. Lymphadenography; a new method for the visualization of enlarged lymph nodes and lymphatic vessels. *Acta radiol.*, May, 1956, *45*, 389–395. (From: Roentgen Department and the Medical Department, Rogaland Sjukehus, Stavanger, Norway.)

A new technique is described for the visualization of lymphatic vessels and lymph nodes following the percutaneous injection of 4–5 cc. of a thin iodized oil into enlarged lymph nodes. The term lymphadenography has been coined to describe the method. A total of 11 cases of lymphadenopathy were subjected to the examination. Visualization of the vessels and lymph nodes was best achieved in cases of leukemia and lymphosarcoma. Visualization was not as successful in metastatic lymphadenopathy since secondary infiltration apparently interferes with flow through the lymph node.

A possible serious complication of this method is the chance of disseminating malignant cells. Slow injection may circumvent this possibility, but further study is indicated. The complication of fat embolism by inadvertent injection into the blood vessels has not been encountered.

The authors suggest that this technique may be useful in determining the effects of therapy on involved lymph nodes. From a therapeutic standpoint

it may be of value in the introduction of radioactive substances into the lymphatic system.—*Michael J. Healy, M.D.*

GENERAL

LUSTED, LEE B., and MILLER, EARL R. An electronic position timer for the fluoroscope. *Radiology*, Aug., 1956, *67*, 259–262. (Address: L. B. Lusted, Department of Radiology, University of California School of Medicine, San Francisco 22, Calif.)

To constantly obtain properly exposed fluoroscopic spot films, the radiologist's mere estimate of required exposure time is generally not reliable. Even though greatly improved, phototimers are expensive complicated devices in which the exposure depends upon the average density of the area of fluorescent screen scanned. If there is a large amount of barium within the scanning area, the background is overexposed.

The authors have devised a simple inexpensive spot-film timer, basing the exposure time upon the thickness of the abdomen of the individual patient. The fluoroscopic table top and the screen mounting are used as a caliper to measure patient thickness. From this relationship, a time scale and indicator were built, and attached to the fluoroscopic apparatus. This was coupled with a simple electronic timer which would automatically adjust the spot-film exposure time to that indicated by the time scale. The thickness of pressure cones is compensated for, and a relay prevents double exposures. The interested reader is referred to the original text for the details of construction. A circuit diagram and several photographs of the equipment are included.

Several other useful features may be added. The basic timer system may be so altered that it will serve for spot films made during chest fluoroscopy. Predetermined spot-film exposure values for specific types of examinations can also be incorporated, and a selector switch will allow the radiologist to choose a variable or predetermined time scale, as the need arises. Since this exposure timer depends upon individual patient thickness, the background density does not vary with the amount of barium in the spot-film area, and for this reason is particularly useful for gastrointestinal studies.—*Charles L. Stevenson, M.D.*

POPPEL, MAXWELL H., and ZEITEL, BERTRAM E. Roentgen manifestations of milk drinker's syndrome. *Radiology*, Aug. 1956, *67*, 195–199. (Address: M. H. Poppel, Department of Radiology, Bellevue Hospital Center, New York 16, N. Y.)

Milk drinker's syndrome was described by Burnett, *et al*, in 1949. It occurs most often in patients being treated for peptic ulcer, with a history of prolonged excessive intake of milk and alkali, with renal insufficiency. Conflicting opinions as to the pathogenesis have since appeared in the literature, and are reviewed here. It is the authors' opinion that during prolonged excessive milk consumption, the blood becomes saturated with calcium and phosphorus, leading to their precipitation in the renal tubules and parenchyma. With alkalosis present, more acid is excreted, resulting in greater precipitation of calcium salts in the kidney. When renal insufficiency occurs, calcium excretion becomes fixed, and the serum levels of both cations remain elevated, resulting in metastatic calcinosis, and more nephrocalcinosis with perpetuation of the cycle. A milk-free diet will return the serum calcium and phosphorus to normal levels, with subsequent reversal of the metastatic calcinosis, but not the renal insufficiency. It is suggested that periodic checks on the serum calcium, NPN, and CO_2-combining power be made in all patients medically managed for peptic ulcer. If abnormal findings develop, milk and absorbable alkali should be eliminated from the treatment regimen.

The roentgenographic demonstration of metastatic calcinosis in a patient with a history of prolonged excessive milk consumption should suggest the possibility of milk drinker's syndrome. These amorphous calcific deposits in the subcutaneous tissues, often near joints, may vary in size from small nodules to large masses. In addition, widespread calcifications in blood vessels, falx cerebri, kidneys, and lungs may be seen. The osseous changes seen in hyperparathyroidism are not present, and the calcium deposition occurs in normal tissues rather than in the dystrophic tissues of patients with scleroderma. —*Charles L. Stevenson, M.D.*

RADIATION THERAPY

FREID, JACOB R., GOLDBERG, HENRY, TENZEL, WILLIAM, OKRAINETZ, CLARA L., and ARAL, M. ISAMETTIN. Cobalt 60 beam therapy: Three years experience at Montefiore Hospital (New York). *Radiology*, Aug., 1956, *67*, 200–209. (Address: J. R. Freid, 1049 Park Ave., New York 28, N. Y.)

The authors present a discussion of their three years' experience in the use of cobalt 60 beam therapy. Aside from the actual results of treatment, a number of practical advantages in the use of this type of therapy are considered.

Radiation in the supervoltage range offers distinct advantages over conventional 200 to 400 kv. radiation for the treatment of cancer. While the cost and maintenance of supervoltage roentgen equipment is prohibitive for small cancer centers, the cobalt 60 beam therapy unit such as is in use at the Montefiore Hospital is a simple, relatively inexpensive, and reliable apparatus for the production of

gamma rays equal in penetration to roentgen rays produced by a 3 mev. machine.

The advantages of supervoltage over conventional radiation are purely physical. Because of increased penetration, the depth dose is greater. Because the tendency to side-scatter is less with cobalt, the volume of normal tissue irradiated is smaller, resulting in less constitutional disturbance. Skin damage is considerably reduced, and absorption in bone is greatly decreased. Cobalt 60 can therefore be used to treat tumor underlying normal bone with less danger of bone necrosis.

The method by which cobalt 60 beam therapy is applied is most important. The services of a radiation physicist are essential, since accurate dosage planning and beam direction are integral parts of treatment. For the radiation to be most effective, with the least damage to healthy tissue, the rays must be so directed that they strike the tumor from several ports of entry.

The majority of patients which were treated with cobalt beam therapy during the time under consideration had advanced malignant disease. Except for isolated instances, pathologic proof of cancer was obtained before treatment had begun. Although the material was poor, in so far as it consisted principally of advanced cancer, results were encouraging in cancer of the lung, the head and neck, and the cervix and body of the uterus. Patients treated for control of symptoms from metastases responded well. The response of tumors of the brain and gastrointestinal tract were generally poor, although symptomatic improvement was marked in some cases of inoperable or recurrent carcinoma of the rectum and rectosigmoid regions. Three patients, considered unfavorable for surgery, received treatment for cancer of the breast. While the results following treatment were somewhat encouraging, tumor cells were found in the breast three to four months after therapy in 2 of the patients, and the third died nine months postirradiation. It is hoped that further study of this problem will yield more satisfactory results.—*A. Ralph Watson, M.D.*

Hsieh, C. L., and Uhlmann, Erich M. Experimental evaluation of the physical characteristics of a 45 MEV medical linear electron accelerator. *Radiology*, Aug., 1956, *67*, 263–272. (Address: C. L. Hsieh, Tumor Clinic, Michael Reese Hospital, Chicago 16, Ill.)

This paper is a highly technical one and does not lend itself to condensation because of the abundance of factual information. The authors' own summary best describes the subject as they covered it and is as follows:

The essential characteristics of high energy external electron beams produced by a traveling-wave linear accelerator were studied experimentally. The principles of design and the performance of the magnet system used for the control of the energy, direction and dimensions of the electron beam are outlined.

Depth-dose curves from electron beams of various energies were obtained, and the effect was studied. Stray radiations associated with the operation of the accelerator were measured for evaluation of safety during therapeutic applications.—*Donald N. Dysart, M.D.*

Notter, Gustaf. Die Behandlung maligner Testistumoren am Radiumhemmet, Stockholm; Bericht über 247 Patienten. (The treatment of malignant testicular tumors at the Radiumhemmet in Stockholm; report on 247 patients.) *Acta radiol.*, June, 1956, *45*, 483–502. (From: Radiumhemmet and Karolinska Sjukhuset, Stockholm, Sweden.)

At the Radiumhemmet in Stockholm, 250 testicular tumors in 247 patients were treated with radiation in the years 1921 to 1954. Thirty-three of them were first seen between 1951 and 1954 and are therefore excluded from the general statistics. In patients with seminoma, the average age, 40.8 (21–80) years, was slightly higher than that of all other forms, calculated at 34.0 (0.5–77) years.

Ninety-seven per cent of the patients had simple orciectomy (the other 3 per cent were inoperable or had more extensive surgery) followed by radiation (given also to the other 3 per cent), using 180 kv., 20 ma, target-skin distance 30–60 cm., 0.5 Cu+1.0 Al (or Thoraeus 0.4 Sn+0.25 Cu+1.0 Al) filter, half-value layer 0.8–1.8 Cu, 50 r/min., and port size 3–400 cm^2. One alternate field was irradiated daily with 300–400 r/skin to a total of 2,400 r/skin per port. In the first years, 2 to 3 series were given within the first 6–7 months. Since tumor doses of 1,500 r are held to suffice for the eradication of seminoma tissue (radiosensitivity being grossly correspondent to the degree of differentiation) one may sometimes succeed to control even cases with extensive metastases. Some of the patients with generalized disease received total body radiation up to 150 r in eight days.

Among the 214 patients, treated between 1921 and 1950, there were 107 (50 per cent) with seminoma, 39 (18.2 per cent) with embryonal carcinoma, 8 (3.8 per cent with mature teratoma, 59 (27.6 per cent) with teratocarcinoma, none with chorionepithelioma, and one (0.4 per cent) with a nongerminal tumor, a mesonephric carcinoma. The five-year survival data for these 214 cases (observed for 5 to 24 years) include 108 (50.5 per cent) symptom free patients, 4 with metastases, and 102 (47.7 per cent) deaths. The five-year "cures" in seminoma were 75 (70.1 per cent), of whom 5 (18.5 per cent) had, and 70 (87.5 per cent) did not have, clinical evidence of metastasis when treatment was started. For other

forms, the five-year "cures" amounted to 5 (12.8 per cent) in embryonal carcinoma, 4 (50 per cent) in mature teratoma, and 24 (42.8 per cent) in teratocarcinoma.

Some discussion is devoted to the possible etiologic relationship between testicular tumors and trauma, cryptorchidism, and hormonal imbalance (12.8 per cent of the 247 patients had a history of undescended testicles). The prognostic value of the sedimentation rate is stressed, stating that a high rate indicates an unfavorable outcome. When the sedimentation rate is low, or becomes low during treatment, the prognostic outlook is much better; other favorable signs are absence of metastases at the beginning of treatment, absence of perforation of the capsule by tumor tissue (histological), average excretion of chorionic gonadotropin, good general condition, and unaltered hemopoiesis.—*E. R. N. Grigg, M.D.*

Murphy, Walter T., and Schmitz, Alfred. The results of re-irradiation in cancer of the cervix. *Radiology*, Sept., 1956, *67*, 378–385. (Address: W. T. Murphy, Roswell Park Memorial Institute, Buffalo 3, N. Y.)

It is commonly considered that if an initial intensive radiation treatment of carcinoma of the cervix fails, further irradiation is contraindicated. This premise is often borne out by disastrous results following injudicious repetition of treatment courses. The authors state, however, that sometimes it is quite rational and feasible to apply a radiation source to a cancer site even though classically intensive irradiation has already been administered. This is said to be particularly justified in those cases where an alternative surgical attack for curative purposes would entail not only an appreciable mortality risk but also a high degree of permanent disability. With these thoughts in mind, a study of cases retreated by radiation methods at the Roswell Park Memorial Institute is presented.

In this study, 46 patients with cancer of the cervix, recurrent or persistent after an intensive radiation regime, were re-irradiated. Of these, 9 were alive and without cancer more than six years following retreatment. Positive biopsies of the retreated disease were obtained in 7 of the 9 surviving patients and in 25 of the 37 patients who died. Retreatment was either by roentgen therapy or radium alone, or by the two combined.

In 9 salvaged cases and in 23 of the 37 fatal cases, disease was limited to one or more of the following areas: cervix, vagina, bladder, rectum, or parametrium (not of the massive or frozen pelvis type). For the 32 patients without massive fixed pelvic or extrapelvic disease, the eventual five-year salvage was 28.1 per cent. For the 24 patients in this group with positive histologic confirmation of recurrence, the eventual salvage was 29.2 per cent.

In the fatal cases, the average survival time after retreatment was thirteen months, the longest period being forty-one months. The average period of survival after the initial course of irradiation was thirty months.

In the 9 surviving patients complications were as follows: Proctitis, 1; cystitis, 1; proctitis and cystitis, 1; rectovaginal fistula, 2. All patients were active and relatively comfortable at the time of the report.

After evaluation of the results of the treatment of these patients the authors reached the conclusion that re-irradiation of a patient with cancer of the cervix which recurs after an initial intensive radiation regime is an indicated and hopeful procedure, especially when pelvic involvement is limited and the patient is otherwise in good physical condition. The end-results, both as to longevity and function, appear to compare favorably with those afforded by present surgical practice. The authors believe that complications, which are calculated risks, may be minimized by the alert application of medical and nursing care.—*A. Ralph Watson, M.D.*

Fletcher, Gilbert H., and Calderon, Roberto. Positioning of pelvic portals for external irradiation in carcinoma of the uterine cervix. *Radiology*, Sept., 1956, *67*, 359–370. (Address: G. H. Fletcher, M. D. Anderson Hospital, Houston 25, Texas.)

Studies were conducted to determine the anatomic accuracy of pelvic portals for external irradiation in carcinoma of the uterine cavity. Tables were compiled for various techniques: (1) opposing split pelvic portals, (2) anterior split portals and two posterior oblique portals and (3) parametrial portals (anterior, sacral and gluteal) with or without weighted loading.

In planning external beam therapy, the anatomic structures which must be considered can be divided into two groups. The first group includes the vagina, the cervix, the paracervical tissues, the body of the uterus, and the medial portions of the broadand uterosacral ligaments. These structures show considerable variation in their relationship to bony structures. In the planning of external beams to cover these tissues a method of projecting them on the anterior and posterior surfaces of the pelvis is required. This was accomplished by the use of a double arm caliper, constructed with one removable graduated arm. This arm was placed in the vagina with the tip against the cervix, and was left in position while films were exposed with the therapy machine under strict treatment conditions. The roentgenograms obtained in this manner furnished a record of the structures included in the beam.

The second group of anatomic structures which must be considered includes the lateral portions of the broad and uterosacral ligaments, and the pelvic wall lymph nodes. This group of structures has a fixed and fairly consistent relationship to the bony pelvis. Films taken with the therapy machine under

treatment conditions permit identification of bony structures which serve as landmarks to determine the adequacy of the fields.

Anterior portals used by the authors were 12 to 14 cm. high and 15 cm. wide with a central lead strip. The sacral fields, 14×5 cm. in size, divided in the center by a lead strip, contribute chiefly to the hypogastric and the common iliac lymph nodes. The lower margin of these portals was usually 2.5 cm. above the sacro-coccygeal joint, with a tilt of 20° toward the feet. Gluteal portals were arranged so that ischial tuberosity, the sciatic notch and the posterio aspect of the pelvic brim were outlined on the film. Separate gluteal portals were always used in this study.

The authors consider anatomic planning essential for adequate and economic treatment of the tumor-bearing areas. The cervix must be localized carefully. Films taken in the treatment positions are an important part of a carefully planned radiation technique.—*A. W. Sommer, M.D.*

Loeffler, R. Kenneth. A simplified method of estimating integral dose in radiotherapeutic practice. *Radiology*, Sept., 1956, *67*, 371–377. (Address: Temple University Hospital, Philadelphia 48, Pa.)

A technique for estimating integral dose which is sufficiently simple that it may be used as a routine for every treatment plan is presented. It is hoped that better correlation of dose and effect may thus be attained. Depth dose data were collected for radiation of half-value layer of 0.5, 3.0, and 14.0 mm. Cu. For the less penetrating radiation the measurements were taken in a Pressdwood phantom 30 cm. square, using a Westinghouse 250 kvcp. machine at a target-surface distance of 4 meters, and corrected to infinite distance by inverse-square factors. The data for the higher quality radiation were obtained with a 2 mev. Van de Graaff generator using a 45 cm. square phantom and a target-to-surface distance of 10 meters. No inverse-square corrections were made in these data.

Using these depth dose data, curves of integral dose with thickness of part were constructed for each quality of radiation, and a curve for 22 mev. radiation was drawn from data published by Laughlin. In essence, the construction of these curves consists of adding the average doses within each centimeter layer down to each depth. For example, for 100 r in air of radiation of half-value layer of 3.0 mm. Cu, the first centimeter averages 139 r, the next 145, and the third 144. Then to 3 cm. depth, there will be 428 gram-roentgens per square centimeter per 100 r in air, or a factor of 3.28 per roentgen. This factor is obtained for each depth, and a curve is drawn from these values. In practice a mechanical integrator is used with the plotted depth dose curve for greater precision.

By multiplying the air dose delivered to a field by the area of that field, and by the thickness factor obtained from the appropriate curve of integral dose with thickness of part, an integral dose may be computed which will be reasonably correct if the absorbing material is of unit density. For heterogeneous media, the errors will be similar to those resulting from the use of standard isodose curves under the same circumstances.—*Harold C. Hamilton, M.D.*

RADIOISOTOPES

Franco, Victor Hugo, and Quina, Mario Gentil. Pneumo-thyroid; a new procedure for determining the mass of the thyroid gland for the radioiodine treatment of hyperthyroidism. *Brit. J. Radiol.*, Aug., 1956, *29*, 434–439. (From: Laboratório de Isótopos, Instituto Portugues de Oncologia, Lisbon, Portugal.)

Radioactive isotope I^{131} is an effective therapeutic agent in the treatment of hyperthyroidism but there are difficulties in calculating the correct individual dosage. The best results can only be obtained if the dosimetry is exact and if account is taken of the existence of several factors of a biologic nature.

The authors present a new technique by which the volume and consequently the mass of the thyroid gland can be determined. With the patient lying on the left side, to reduce the danger of gas embolism, oxygen is injected in the median line of the infrahyoid region, 1 cm. above the upper margin of the sternum. A 0.7 mm. needle is introduced and it is essential that its point traverses the deep fascia. If uncertain of the passage of the needle through the deep fascia, the patient is made to swallow, and if the needle does not follow the movement of the larynx it is inserted more deeply until it does so. One hundred and fifty cc. of oxygen is injected with a pressure from 2 to 5 cm. of mercury. Immediately after this anteroposterior and lateral roentgenograms and an anteroposterior laminagram are obtained.

The method has proven to be safe but it has not been employed in patients with evident signs of cardiac insufficiency. Using the formula of the volume of scalene ellipsoid an error of over 10 per cent has not been observed in any case which was subsequently operated upon. Visualization of the gland has sometimes proven difficult in recurrent postoperative hyperthyroidism owing to the scar. In these cases it may be necessary to perform two injections, one under the scar and one higher up. The results of therapy confirm the importance of the correct assessment of the thyroid gland mass to more accurate dosimetry.—*Arthur E. Childe, M.D.*

Madell, Samuel H., Kligerman, Morton M., Quimby, Edith H., and Fertig, John W. Statistical appraisal of the use of radio-

active iodinated human serum albumin for the detection of liver metastases. *Radiology*, Aug., 1956, *67*, 210–217. (Address: S. H. Madell, 622 West 168th St., New York 32, N. Y.)

The authors explored the possibility of determining the presence or absence of liver metastases by scintillation counter measurements of the differential concentrations of I^{131} HSA using statistical methods. The technique described by Stirrett, Yuhl, and Libby was followed closely.

Each patient received an intravenous injection of 300 microcuries of I^{131} HSA. Twenty-four and forty-eight hours after the injection the counting procedure was performed. Each patient received 10 drops of Lugol's solution three days before the study to minimize the uptake of radioactive material by the thyroid. The distribution of the I^{131} HSA was observed in 52 patients with and without suspected malignant disease. Eight were excluded from the series, leaving 44 surgically verified cases for analysis.

Forty-two points, spaced 5 cm. apart, were marked on the torso of each patient through a perforated guide. The count over each of the 42 points was recorded in terms of counts per minute above the background count. The values at individual points were then expressed as percentages of the mean value, which served as a reference. An *in vitro* experiment showed that the measuring procedure was adequate to detect localized concentrations of radioactive material, if such concentration was as much as three times the normal for the point.

Results are tabulated and their significance from a statistical standpoint is discussed. It is concluded that, even when the strictest criteria are imposed for the definition of an abnormal point, it is still not possible to separate the group with liver metastases from the group without liver metastases when intra-abdominal cancer is present.—*A. W. Sommer, M.D.*

SEIDLIN, S. M., SIEGEL, EDWARD, YALOW, A. AARON, and MELAMED, S. Acute myeloid leukemia following prolonged iodine-131 therapy for metastatic thyroid carcinoma. *Science*, May 4, 1956, *123*, 800–801. (From: Medical Physics Laboratory, Medical Division, Montefiore Hospital, New York, N. Y.)

Two instances of acute leukemia developed in a series of 16 patients with metastatic thyroid carcinoma who were treated intensively with radioiodine.

The first patient, a sixty-two year old man, received 13 therapeutic doses of I^{131} totalling 1,455 mc over a four year period. The authors estimate that he received a cumulative blood radiation dose of about 600 rad. He died in the fourth year of therapy with a clinical picture of acute myeloid leukemia. Postmortem examination revealed anaplastic carcinoma of the thyroid metastasizing to cervical lymph nodes, skull, spine and lungs, in addition to myeloid leukemia involving bone marrow, spleen, liver and lymph nodes. The second patient was a sixty-six year old woman who died of acute leukemia one year after receiving 1,730 mc of I^{131} over a five year period. The estimated dose to the blood was 550 rad. The autopsy findings included acute myeloid leukemia with infiltrates in bone marrow, spleen, liver, lungs, kidneys, and pancreas and metastatic thyroid adenocarcinoma in the skull.

A causal relationship between radioiodine therapy and leukemia is not definitely established but a correlation is suggested. In addition, the experimental production of leukemia in animals by radiation, a higher incidence of leukemia among radiologists as compared to other physicians, and the high incidence of myeloid leukemia in the Hiroshima and Nagasaki survivors are all consistent with the existence of a relationship between the radiation received during massive radioiodine therapy and the subsequent development of leukemia.

The body radiation dose received during I^{131} therapy for hyperthyroidism is, at most, a few per cent of that received by those treated for thyroid carcinoma and is considerably smaller than the apparent minimum leukemogenic dose. It is therefore unlikely that the incidence of leukemia in patients treated for hyperthyroidism with radioiodine will be significantly higher than the occurrence of leukemia in the general population.—*John F. Weigen, M.D.*

MCCONAHEY, WILLIAM M., OWEN, CHARLES A., JR., and KEATING, F. RAYMOND, JR. A clinical appraisal of radioiodine tests of thyroid function. *J. Clin. Endocrinol. & Metabol.*, June, 1956, *16*, 724–734. (From: Section of Medicine and Section of Clinical Pathology, Mayo Clinic, and Mayo Foundation, Rochester, Minn.)

The relative merits of 7 tests for the evaluation of thyroid status were studied in 389 patients. Of these patients, 181 were euthyroid controls, 38 were initially suspected of being hyperthyroid, but were found to be uthyroid, 92 had exophthalmic goiter, 46 had adenomatous goiter without hyperthyroidism, 19 had adenomatous goiter with hyperthyroidism, and 13 patients had myxedema. The diagnosis in each patient was independent of I^{131} tests and was based on clinical evaluation and tests such as BMR and serum PBI. Comparisons were made of the percentages of the 6 hour thyroid uptake, 24 hour thyroid uptake, and 24 hour urinary excretion, the per cent per hour accumulation rate, per cent per hour extrarenal disposal rate, 24 hour conversion ratio, and ml./min. thyroid iodide clearance. BMR and PBI determinations were performed in these patients as well.

The 6 hour thyroid uptake and the thyroid iodide

clearance rate were the most precise tests for separating hyperthyroid and euthyroid patients. Of the two, the 6 hour uptake was superior for large scale use because of its relative technical simplicity. Urinary excretion, accumulation rate, and extrarenal disposal rate tests are complex and, because they require precise patient cooperation, are sometimes inaccurate. The conversion ratio is accurate, but requires a larger tracer dose than is warranted for routine use.

The 6 hour thyroid uptake is as accurate as the 24 hour uptake and in some instances may be more accurate, in addition to being simpler and quicker than the 24 hour uptake.

Only the 6 hour and 24 hour thyroid uptakes were valuable in the diagnosis of myxedema and none of the tests were conclusive in determining whether or not a patient with a nodular goiter had hyperthyroidism.—*George Roberts, M.D.*

Jaimet, C. H., and Thode, H. G. Further clinical studies of thyroid and salivary gland function with radioiodine. *Canad. M.A.J.*, June, 1956, *74*, 865–870. (From: Department of Medical Research, Hamilton College, McMaster University, Hamilton, Ontario.)

Previous reports by the authors indicated a close correlation between thyroid function and the secretion of iodine by the salivary glands. In the present study, tests were performed on 350 patients in which 24 hour saliva activity was compared with the 24 hour thyroid uptake, conversion ratio, plasma I^{131} activity, and PBI^{131}. Two hundred twenty of these patients were normal controls, 70 were hyperthyroid, 40 were hypothyroid, and 20 had had prior I^{131} therapy; the diagnosis was based on clinical evaluation, thyroid uptake, and conversion ratio.

The saliva-PBI^{131} ratio was judged to be almost 100 per cent accurate and was of particular value in the diagnosis of hypothyroidism; this was confirmed in each hypothyroid patient by use of the TSH test. The values for the saliva-PBI^{131} activity ratio are: hyperthyroid—0.14 to 44; euthyroid—150 to 250; hypothyroid—400 to 1,900 or over with the borderline ranges from 50 to 150 and 250 to 400 for mildly hyperthyroid and hypothyroid patients respectively, as well as euthyroids.

The saliva-PBI^{131} ratio has good correlation with the thyroid uptake and conversion ratio and appears to follow more closely the clinical symptoms of patients being treated with I^{131}, propyl thiouracil, or other therapy.

A discussion of the role of the salivary glands in iodine metabolism is presented.—*George Roberts, M.D.*

Wetherley-Mein, G., Hutt, M. S. R., Langmead, W. A., and Hill, M. J. Radioactive iron studies in routine haematological practice. *Brit. M. J.*, June 23, 1956, *1*, 1445–1449. (From: Louis Jenner Laboratory and Department of Physics, St. Thomas's Hospital and Medical School, London, England.)

This paper reports the use in hematologic laboratory tests of Fe^{59}, obtained from Oak Ridge through the Harwell (England) Isotope Division. The radioactive material has been injected either combined with beta-globulin in a buffered solution, or added to 0.1 per cent sodium citrate in isotonic saline; in both cases 13 ml. of solution containing 13 μc were prepared, and 10 ml. of it injected, the remainder being retained for counting and calculation of the total injected dose.

Plasma clearance can be determined in the well counter from samples of venous blood, taken at 15, 25, 40, and 60 minutes after the injection. *Surface counting* is done with a directional scintillation counter which reveals changes in activity over the precordial region, spleen, liver, and sacrum (marrow activity); these measurements are continued for ten days after the injection, and the results are plotted on linear ordinates against time on a log abscissa. *Red-cell utilization of injected iron* can be calculated from: (a) the red blood cell volume, estimated from the patient's weight and hematocrit; (b) the total injected dose in counts/sec.; and (c) the activity in counts/sec./ml. of red cells, by using the formula $(ac \times 100)/b$.

In the "normal" subject, the injected iron is cleared from the plasma exponentially, with a half-clearance of 70–120 minutes. The surface counts over the heart fall rapidly in the first 2–3 hours, while the counts over the sacrum begin to rise, and reach a peak at 20–30 hours. Red cells containing active hemoglobin appear in the peripheral blood after about 10 hours, and increase in number up to ten days. The liver shows a slight initial rise, followed by a gradual fall, while the spleen follows the changes over the heart. Seventy to eighty per cent of the injected dose eventually appears in red cells, its maximum level being reached in 7–14 days.

Seventy patients have been investigated. The results revealed 3 distinctive patterns: (1) no significant changes were found in leukemia, uremia, and malignant lymphoma; (2) rapid plasma clearance, and high utilization of Fe^{59} for hemoglobin synthesis, without notable alterations of the marrow pattern, were seen in iron deficiency anemia, posthemorrhagic anemia, polycythemia, and some "refractory" anemias; and (3) slow plasma clearance, and low percentage utilization, with flat marrow curve and gradually rising liver uptake (suggesting depressed erythropoiesis), were commonly observed in aplastic anemia, but sometimes also in "refractory" anemia, in leukemias with low hemoglobin, and in myelofibrosis.

Five cases are reported to illustrate the details of the procedure. The authors believe that the radia-

tion hazard to the patient is negligible, and the risk to the staff even less, provided simple precautions are observed. The techniques are not difficult, and well within the scope of a routine laboratory of a hematology department. Although the procedure is not deemed to be indispensable to diagnosis and treatment of the majority of patients with blood disorders, it can demonstrate objectively erythropoietic abnormalities, and is of help in the stu y of the dynamics of erythropoiesis in any given case. Radioiron tracer studies are therefore considered as having a definite place in the routine investigation of hematologic conditions.—*E. R. N. Grigg, M.D.*

KEREIAKES, J. G., and KREBS, A. T. X-radiography with beta-emitting isotopes. *Radiology*, Sept., 1956, *67*, 419–421. (Address: J. G. Kereiakes, Radiobiology Department, Army Medical Research Laboratory, Fort Knox, Ky.)

The authors discuss the use of beta-emitting isotopes in obtaining radiographs. The material used in this experimental study consisted of a 300 mc source of S^{90}–Y^{90}. This source in its normal form emits x-rays resluting primarily from *bremsstrahlung* and characteristic x-radation produced in the source and absorber material. It is this x-radiation which was used for these radiographic studies.

Radiographs of a rat body and of a hand show rather good definition and contrast in spite of the large "focal spot" of the source (5 mm. in diameter) and the relatively short source-specimen distance (about 5 inches).

It is thought that better radiographs can be obtained by the use of smaller active diameter sources and with other beta-emitting isotopes and/or target materials emitting radiations properly fitted to the requirements of the body parts to be radiographed. —*Donald N. Dysart, M.D.*

MISCELLANEOUS

MILLER, C. E., and MARINELLI, L. D. Gamma-ray activity of contemporary man. *Science*, July 20, 1956, *124*, 122–123. (From: Radiological Physics Division, Argonne National Laboratory, Lemont, Ill.)

The predominant activity in a number of uncontaminated subjects is found to be K^{40} to the extent of about 0.19 per cent of body weight in men and 0.15 per cent in women; these values are in good agreement with those found by isotope dilution methods. The presence of Cs^{137} was also detected but the activity, about 10^{-9} c, is considerably lower than accepted permissible levels. The presence of this isotope appears to be a result of deposition on grazing lands from nuclear fall-out. The absence of other fission products found in air and dust indicates their low retention in the body. Radium, with an expected activity of 0.5×10^{-10} c, could not be measured in the presence of the potassium. Technical details of the method of measurement are included.—*Raymond S. Quick, B.S.*

SPIERS, F. W. Radioactivity in man and his environment; Presidential Address. *Brit. J. Radiol.*, Aug., 1956, *29*, 409–417. (From: Department of Medical Physics, The University of Leeds, Leeds, England.)

The natural background of radiation to which man is inevitably exposed during life provides a lower, tolerable level of dose rate as a basis for comparison in attempting to assess permissible amounts of added artificial radiation. Natural radiation is due to external sources, principally the gamma rays from terrestrial radioactivity and cosmic rays and internally from the alpha and beta activity of body tissues and of air, food and water. The order of radioactivity and the typical levels of radiation resulting from these different sources are summarized in the following table.

	γ ray equivalent (g. Ra per g.)	Radiation background (mrad per year)
Rocks	$1–12\times10^{-12}$	Calc.: 26–228 Meas.: 80–100* 50†
Seawater	3×10^{-14}	0.5
River water	10^{-16}	0.001
	Radon, c per l.	
Open air	$0.3–4.3\times10^{-13}$	4.0
Indoors	$0.8–9.1\times10^{-13}$	
	g. Ra per g.	
Water	$0.1–23\times10^{-16}$	
Food	$0.04–125\times10^{-16}$	
Body: K^{40}	13×10^{-9} c	20
C^{14}	87×10^{-9} c	1
Ra	$0.05–0.3\times10^{-9}$ g.	2
Cosmic rays		28

* Indoors.
† Outdoors.

The terrestrial component of background radiation is due mainly to the U and Th series and in Swedish alum shales the high U content increases the gamma equivalent to about 60×10^{-12} g. Ra per g. A value of 3×10^{-13} c per l. is regarded as an average estimate for the radon content of air but, inside buildings, this will be influenced by ventilation and the constructional materials and will generally be higher than the corresponding open-air value; the level in the Joachimsthal mines may be 10,000 times higher. Similarly the radium content of thermal springs is of the order of $1{,}000\times10^{-16}$ g. per g. but there appears to be little correlation between the activity of

normal drinking waters and body radioactivity, indicating that the important source of intake is via foods.

While the external gamma radiation background is uniform for the whole body, except for the screening effect of overlying tissues, the internal alpha and beta radiation dosage is a localized effect dependent on the radioactive content of the particular tissue. In view of the low dose rates due to natural activity, long term genetic and carcinogenic changes are most significant. The following annual doses are calculated for average estimates of the radiation levels:

Gonads and soft tissue	100 mrad.
Lungs	200–400 mrem.
Bone	120 mrem.

Variations in gonadal dose rates from 90–300 mrad per year are found for differetn localites.

In comparison, the ingestion of 0.6 μc of Cs^{137} and $10\text{-}45 \times 10^{-9}$ c of Sr^{90} would be required to double the already existing radiation dose rate of the body. These fission products are of significance because of their long life and the possibility of their being taken up by the body.—*Raymond S. Quick, B.S.*

Van Middlesworth, L. Radioactivity in thyroid glands following nuclear weapons tests. *Science*, June 1, 1956, *123*, 982–983. (From: Department of Physiology, University of Tennessee, Memphis, Tenn.)

The radioactivity of thyroid glands, considered as due to I^{131}, has been measured for 175 human autopsy specimens and 1,044 cattle from the Memphis, Tennessee, area over the period of October, 1954 through March, 1956. Similar measurements are reported on sheep and cattle thyroids from Washington State, Canada, England, Germany and Japan between March, 1955 and February, 1956.

The data for Memphis cattle show three periods of increased activity, two due apparently to non U.S.A.E.C. nuclear tests and a main peak with a maximum of 10,000 times the normal minimum activity following a series of Nevada and Pacific tests in 1955. There was an interval of about five days between the beginning of these tests and a detectable increase in I^{131} activity, and fourteen to twenty days before the maximum was reached, the activity remained above the minimum for about eight months. Similar variations in activity were found for sheep from England and Germany but occurred two to four weeks later.

The maximum thyroid I^{131} content per gram for humans was less than 0.5 per cent of that for cattle, which was in turn consistently lower than for sheep from the same area. For the Memphis cattle the calculated average dose to the thyroid was 0.11, 4.3 and 0.04 rep. for the three periods of fall-out and for the English cattle and sheep 0.15 and 0.4 rep. between April and November 1955, in comparison with the 5–10,000 rep. required for the treatment of hyperthyroidism in humans.

The method and accuracy of measurement are outlined and the factors influencing the distribution and accumulation of I^{131} following nuclear tests are discussed.—*Raymond S. Quick, B.S.*

Sacher, George A. On the statistical nature of mortality, with especial reference to chronic radiation mortality. *Radiology*, Aug., 1956, *67*, 250–258. (Address: Argonne National Laboratory, Box 299, Lemont, Ill.)

The theory is presented that death is a consequence of the dynamics of physiologic processes. Its occurrence at a particular time in a particular animal is attributed to the prior occurrence of a rare fluctuation of large amplitude in the physiologic state of the animal. By means of a simple mathematical model of a physiologic fluctuation process, it can be shown that the probability of occurrence of such fluctuations is a function of the values of certain vital physiologic parameters. An approximate relation between rate of mortality and mean physiologic state is derived, which indicates that the logarithm of the rate of mortality is a linear function of the mean physiologic state of a population. Application of this relation to chronic mortality following various patterns of radiation exposure yields results that are quantitatively consistent and in accord with present knowledge about permanent radiation injury.

It is stated that the dose-dependence of the permanent damage is strikingly reminiscent of the dose-dependence found for induced chromosome aberrations. Muller and Lea have given full discussions of the genetic considerations that led them to conclude that permanent radiation injury in mammals must be due primarily to chromosomal aberrations. In view of the indirect evidence presented here, it becomes increasingly important that the role of chromosome damage in late effects be investigated cytologically.

According to the author, the developments presented here are still far from adequate for the task of prediction that was assigned at the beginning. An attempt has been made to present some concepts that are felt to be fundamental to an understanding of the factors that govern the stability of living organisms. The logical clarification and experimental implementation of these concepts will, however, require much additional work.—*A. Ralph Watson, M.D.*

Spiess, Heinz. Schwere Strahlenschäden nach der Peteosthorbehandlung von Kindern. (Serious radiation damages following peteosthor-therapy of children.) *Deutsche med. Wchnschr.*, June, 1956, *81*, 1053–1054. (Address: Univ.-Kinderklinik, Kirchweg 38, Göttingen, Germany.)

Peteosthor is a mixture of thorium-X with platinsol and red eosin (peteos). The latter two ingredients are supposed to be a vehicle for a selective storage of the alpha rays of thorium-X in the diseased tissues. The drug has been used mainly in extrapulmonary forms of tuberculosis.

Experimentally, it has been proven that the drug is not stored selectively in the diseased tissues, but rather in the osseous skeleton. Detrimental results have been noted in the hematopoietic system, the metabolism, the kidneys, the liver and genetically. The growth of children and adolescents has been distinctly retarded and malignant neoplasms are known to have developed four to seven years after the therapy.

In a series of 49 cases, 3 died prematurely from the original tuberculosis and a thorium-induced leukemia. In 11 cases malignant neoplastic changes developed with 9 fatalities and only 2 survivals.

Experimentally, it has been proven that in the average case the skeleton receives 5 to 10 times the tolerance dose of alpha rays of thorium-X and all other tissues a corresponding overdose. The author concludes that according to his follow-up studies the drug has proved hazardous and ineffective.—*Ernest Kraft, M.D.*

JANES, JOSEPH M., HIGGINS, GEORGE M., and HERRICK, J. F. The influence of splenectomy on the induction of osteogenic sarcoma in rabbits. *J. Bone & Joint Surg.*, July, 1956, *38-A*, 809–816. (From: Section of Orthopaedic Surgery, Section of Anatomy, and Section of Biophysics, Mayo Clinic and Mayo Foundation, Rochester, Minn.)

Osteogenic sarcoma develops in 50 per cent of rabbits given intravenous injections of beryllium silicate. It was found that in all animals in which tumors developed there were atrophic changes in the spleen, whereas in those animals in which tumors did not develop the spleens were functional and hyperplastic. In animals developing tumors the histiocytes of the spleen phagocytized the beryllium and were destroyed; new histiocytes were formed from the reticuloendothelial components and were destroyed, so that functioning splenic tissue degenerated and became avascular.

The spleen has been shown to have a protective role against ionizing radiation. In mice when the spleen is removed and total body radiation given the incidence of lymphoid tumors is increased. When the spleen is shielded or implanted into the peritoneal space its protective role is shown. Numerous experiments have demonstrated some humoral mechanism elaborated by the spleens of mice and dogs.

Eleven rabbits received beryllium injections after splenectomy. Three died from unrelated causes before the length of time necessary to develop bone tumor. Of the other 8, all but 1 developed osteogenic sarcoma and that one had bone marrow changes interpreted as being the earliest stage in the development of osteogenic sarcoma.

Apparently the spleen tends to provide protection in the rabbit against development of beryllium induced osteogenic sarcoma.—*Martha Mottram, M.D.*

GUÉRIN, MAURICE. Influence des rayons x sur l'apparition d'adénocarcinomes mammaires chez des souris inoculées avec de telles tumeurs contenant des corps d'inclusion. (Influence of x-rays on the development of mammary adenocarcinomas of mice inoculated with tumors containing inclusion bodies.) *Bull. Assoc. franç. l'étude cancer*, Jan.–March, 1956, *43*, 23–36. (From: Laboratoire de Médecine expérimentale and Institut de Recherches sur le Cancer Gustave-Roussy, Paris, France.)

The article presents details of experimental work on mice of different strains in an attempt to evaluate the influence of roentgen rays (total body irradiation of 300 r as measured at the top of the metallic wire cage, at 200 kv., 1 mm. Cu and 2 mm. Al filtration, 65 cm. distance). As to the development of mammary adenocarcinoma, the author analyses the findings as well as results of other experiments and brings forth suggestions of value. Variations of the strains of mice in their susceptibility to mammary adenocarcinoma are borne out clearly, indicating significance of herdity. Thus mice of Swiss strain, hereditary line E were remarkably refractory and out of 40 irradiated, only 1 developed a mammary tumor, whereas out of the control group (not irradiated) of 80, none showed the tumor.

Total body roentgen irradiation seems to have an inhibitory effect on the development of mammary tumors if applied before puberty, whereas if applied after puberty, the action is a stimulating one, indicating an influence of hormonal factors.

The author investigated the lactogenic hormonal factor and found it to be of no significance. In contrast, the tumor incidence in ovaries of mice irradiated after puberty was rather high. The incidence of mammary adenocarcinoma was in turn quite high in those mice showing ovarian tumors with hormonal activity.

The increase in percentage of inclusion bodies (in tumoral tissue extract injected to mice, both irradiated and nonirradiated) does not seem to increase the incidence of mammary tumors. These inclusion bodies do not, therefore, represent an active form of the virus (Bittner's), which in turn does not seem to become reactivated by total body irradiation. The increase in the incidence of mammary adenocarcinoma of mice seems to be due to a hormonal disbalance in which the ovary apparently plays the important role.—*Jirair N. Sarian, M.D.*